The association between paediatric traumatic brain injury and antisocial behaviour in adulthood: a longitudinal study using the ALSPAC data

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Volume I: Systematic Literature Review and Empirical Research Project

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Systematic Literature Review

The association between paediatric traumatic brain injury and antisocial behaviour: a systematic review of the literature.

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Abstract

Despite growing evidence supporting a link between paediatric traumatic brain injury (TBI) and engagement in antisocial behaviour, few studies have taken a rigorous approach in evaluating this. The present review systematically explored previous literature examining the association between TBI before the age of nineteen years old and engagement in severe behavioural problems such as for instance violence, aggression and assault. All articles published from 1990 to 2016 were searched using four major databases (Ovid MEDLINE, PsycINFO, Embase, Web of Science), alongside manual searching and cross-referencing. The level and quality of evidence were evaluated using quality assessment tools selected from previous literature. A total of 14 studies were found to meet eligibility criteria. Taken together, they supported the presence of an association between paediatric TBI and antisocial behaviour, and identified some potentially intersecting factors (e.g., emotional dysregulation, drug and alcohol abuse). However, the studies also consistently presented with a number of methodological limitations, such as, for instance, unclear temporal ordering of TBI and antisocial behaviour; limited information about participants' pre-injury backgrounds; over-reliance on self-report measures. These make it difficult to make meaningful comparisons across studies and draw definite regarding the directionality of the relationship between TBI and antisocial behaviour, and the mechanisms underpinning this association. The findings indicate that there is a need for more extensive and methodologically sound research on the topic. A novel, age-graded theoretical model examining the relationship between paediatric TBI, antisocial behaviour, and different child- and parent-based risk factors was introduced, before this is described in more detail and tested in the next chapter of the present thesis. The implications of the present systematic review for informing rehabilitation and preventative measures are discussed.

Introduction

Traumatic brain injury (TBI) has been estimated to affect approximately 10 million people every year, and to be one of the leading causes of disability and death in children and young adults worldwide (Dinsmore, 2013). The consequences of TBI with respect to everyday psychological functioning are wide-ranging; these can often include short- and long-term negative changes in cognition (e.g., reasoning, problem-solving), communication skills, emotion recognition and regulation, and behaviour, such as increased irritability, impulsivity, and propensity towards aggression and violence (Langlois et al., 2006; Schretlen et al., 2003).

An area of particular interest is the impact of paediatric TBI on later development. Compared to the adult brain, the child and adolescent brain is thought to be uniquely vulnerable to external insults, and thus at high risk of long-term impairments in the development of key processes responsible for cognitive and emotional regulation (Blanchard et al., 2003). It is therefore unsurprising that paediatric TBI has been linked to several adverse outcomes later in life, such as, for instance, lower educational attainment (Sariaslan et al., 2016), increased rates of substance abuse (McKinlay et al., 2014), internalising (e.g., depression; Bloom et al., 2001) and externalising conditions (e.g., ADHD; Schachar et al., 2004). With respect to the social domain, there is tentative evidence that, within community populations, children and adolescents with history of TBI tend to show less sophisticated interpersonal skills (e.g., Ganesalingam et al., 2007; Gerring et al., 2009), lower levels of prosocial behaviour, and higher levels of aggression and interpersonal violence compared to those who did not suffer from a TBI (Cole et al., 2008; Stoddard & Zimmerman, 2011).

The existence of a possible link between paediatric TBI and engagement in antisocial behaviour, an umbrella term encompassing nuisance behaviour, intimidation and vandalism, has recently become a topic of pressing concern, due to its wider societal implications. In England and Wales, antisocial behaviour has been estimated to cost £3.4 billion per year, and is thus a burgeoning political and public health concern (Great Britain Home Office, 2004). Despite growing recognition that early TBI might be linked to engagement in antisocial behaviour later on in life, few studies have taken a systematic approach in evaluating the evidence for this. A number of previous systematic reviews have focused on cross-sectional studies with incarcerated individuals, showing that there are remarkably high rates of TBI history in this population (see e.g., Allely, 2016, Farrer & Hedges, 2011 for recent reviews), and that offenders with TBI tend to enter the criminal justice earlier and have a higher number of convictions than those without a history of TBI (Williams et al., 2010; Perron & Howard, 2008). However, in these studies participants had

suffered from TBI at different times in their lives, not exclusively during childhood; therefore, the trajectories for those who presented specifically with paediatric TBI remain unclear. Moreover, although these findings were suggestive of a causal relationship between TBI and problem behaviours, since these focused exclusively on prisoner populations, it cannot be ruled out that other mechanisms may be at work. For instance, it is possible that TBI and offending may have other common underlying determinants (Parsonage, 2016).

Studies following community populations, especially those with longitudinal designs, may be more appropriate for elucidating the aetiology of the relationship between paediatric TBI and problem behaviours. Previous reviews examining these types of studies have predominantly focused on examining the link between TBI a broader range of behavioural outcomes, such as psychopathology, social skills and internalising and externalising conditions (see e.g., Anderson et al., 2009; Emery et al., 2016; Li & Liu, 2012); there is no review, to the best of our knowledge, focusing specifically on how TBI might lead to more severe interpersonal problems such as antisocial behaviour. It is also worth noting that several different factors, such as for instance lower socio-economic status (Amram et al., 2015; Piotrowska et al., 2015), substance and alcohol abuse (Bjork & Grant, 2009; Robins, 1998), and emotional dysregulation (Aboulafia-Brakha, Allain, & Ptak, 2016; Davidson, Putnam, & Larson, 2000) have been linked to both TBI and emergence of problem behaviour; nonetheless whether and how such variables intersect the relationship between paediatric TBI and antisocial acts has yet to be combined and evaluated as a whole.

It is therefore critical to examine systematically the recent empirical evidence to provide a comprehensive overview of the ongoing progress in the area. The strong need for more attention towards this area of research has been recently highlighted by a number of reports within the UK (Parsonage, 2016; The British Psychological Society, 2015; Williams, 2012). The Office of the Children Commissioner, a national organisation led by the Children's Commissioner for England, which aims to promote and safeguard the views and interests of all children in England, highlighted how there are currently significant percentages of young people living in custody within secure settings in the UK who might have undiagnosed or untreated neurological issues (Hughes et al., 2012); this has led to the comparison of TBI within offending populations to a "silent epidemic" (Williams, 2012). It is essential that more time and effort are dedicated to increase of our understanding of the consequences of paediatric brain injury and whether these might contribute or explain offending and other criminal behaviours. This might in turn support the development

of measures and interventions to prevent or minimise the impact of antisocial behaviour at individual, societal and financial level.

In summary, there is growing recognition that paediatric TBI may be an important precursor of antisocial behaviour, although little work has examined the empirical evidence for this using a systematic approach. Expanding our knowledge of the relationship between such variables has important implications for informing preventative and early intervention measures. Based on this, the present systematic literature review thus aimed to address the following research questions:
I) Is paediatric TBI associated with increased engagement in antisocial behaviour later on in life?
II) What factors have been found to intersect the association between paediatric TBI and antisocial behaviour?

Method

The systematic literature review described in the present study was carried out according to the PRISMA guidelines (available here: www.prisma-statement.org; Moher et al., 2009). The eligibility criteria used to determine which studies were going to be included in the review, the search sources and strategy, and the study selection and quality assessment processes will now be described.

3.1 Eligibility criteria

To be included in the systematic review, studies had to meet the following criteria: (1) the study populations were human participants; (2) at least one of the study groups had sustained a TBI before nineteen years of age; (3) studies had been published as original articles in peer-reviewed journals; (4) they were written in English; (4) TBI and antisocial behaviour were conceptualised as described in 3.1.1 below; (5) the articles had been published from 1990 to December 2016. 1990 was selected as a cut-off for similar reasons as those mentioned by previous reviews on the outcomes of paediatric TBI (e.g., Li & Liu, 2012), i.e. due to greater interest in the area following this year, and also to maximise the likelihood of overlap in the methodologies and outcome measures used by the studies included in the review. Unpublished dissertations, conference proceedings, abstracts without locatable full texts, review articles and intervention studies were all excluded. Studies which did not provide with information regarding participants' age at TBI were also not included.

All decisions regarding inclusion and exclusion were made by at least two researchers, always including at least one senior researcher. When there were disagreements between two researchers that could not be resolved, studies were further reviewed by a third, senior researcher.

3.1.1 Definitions

In the present review, TBI and antisocial behaviour were conceptualised in the following ways.

3.1.1.1 Definition of Traumatic Brain Injury

Definitions of TBI and classifications (e.g., according to type, severity, or mechanism) vary widely across studies, specialities and countries (Thurman, Coronado, & Selassie, 2007). This is considered a significant current pitfall of data gathering in TBI research, and possibly the largest obstacle with respect to evaluating the validity and robustness of published literature and interventions (Haydel, 2016; Saatman et al. 2008). In the present review, definitions provided by the selected articles needed to be consistent, as an absolute minimum, with the broader definition provided by the Centres for Disease Control and Prevention (2014; also adopted by previous systematic reviews on TBI, e.g. Hughes et al., 2015). This operationalises TBI as "a bump, blow, or jolt to the head or a penetrating head injury that disrupts the normal function of the brain". It was decided not to adopt more stringent criteria for defining TBI in order to maximise the number of studies included in the review.

3.1.1.2 Definition of antisocial behaviour

Similarly to TBI, antisocial behaviour is also a relatively heterogeneous concept, with no single definition (Carr & Cowan, 2006). Typically, it refers to a wide spectrum of activities considered unacceptable by one's cultural standards, and disrespectful of other people's rights (Frick, 1998). Specific labels, classification systems, and assessments methods vary depending on several factors, such as for example discipline (e.g., psychology, public health, criminology), context, and country; this has led to difficulties for reviews and meta-analysis studies with respect to summarising current evidence in this area (Rhee & Waldman, 2002). For the purpose of the present review, the term "antisocial behaviour" was used to refer to activities on the most severe end of the spectrum of socially unacceptable behaviour, such as for instance: rule-breaking, delinquency, nuisance behaviour, vandalism, and physical and verbal aggression. Studies focusing exclusively on less severe types of behavioural difficulties (such as e.g., externalising behaviours or conduct problems) were thus excluded.

3.2 Search sources and strategy

The searches for the present review were conducted using the following search engines: Ovid MEDLINE, PsycINFO, Embase, and Web of Science. The final search strategy was developed following examination of both published and prospective systematic reviews and meta-analyses investigating paediatric TBI and behavioural problems. Three key concepts were identified for the search: TBI, childhood, and antisocial behaviour. Terms and synonyms relating to each of these

were combined using Boolean operators. Limiters and filters were used to apply the inclusion and exclusion criteria described in 3.1 above. Manual searching for additional manuscripts was also conducted by consulting reference lists and previous systematic reviews and meta-analyses on the topic of paediatric TBI (see Appendix A for the detailed search strategies used for each database).

3.3 Quality assessment

The information gathered from the final selection of studies (including: sample characteristics, study design, methodology, instruments used, and robustness of main findings) was assessed for methodological quality and risk of bias. It should be noted that there are currently no recognised "gold standard" tools for the quality assessment of either cross-sectional or longitudinal studies. An investigation of previous evidence suggests that the NIH Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies (US Department of Health, 2013; https://www.nhlbi.nih.gov/health-pro/guidelines/in-develop/cardiovascular-risk-reduction/tools/cohort) and the Cohort Study Checklist (Critical Appraisal Skills Programme, 2016, http://media.wix.com/ugd/dded87_e37a4ab637fe46a0869f9f977dacf134.pdf) are commonly used tools for the evaluation of cross-sectional and longitudinal studies respectively. These were therefore adopted in the present systematic review. Each study was evaluated by two independent researchers, with an agreement rate of 71%. All disagreements were resolved through discussion.

Results

4.1. Information extraction

4.1.1 Study selection

The initial search from all four databases yielded a total of 2189 studies. 16 additional studies were identified via manual searching, resulting in a total of 2205 potentially eligible studies. Removal of duplicate records reduced the number of potentially relevant studies to 2010. Subsequent selection of studies involved four main, consecutive phases (see Figure 1 for a pictorial summary of the study selection process):

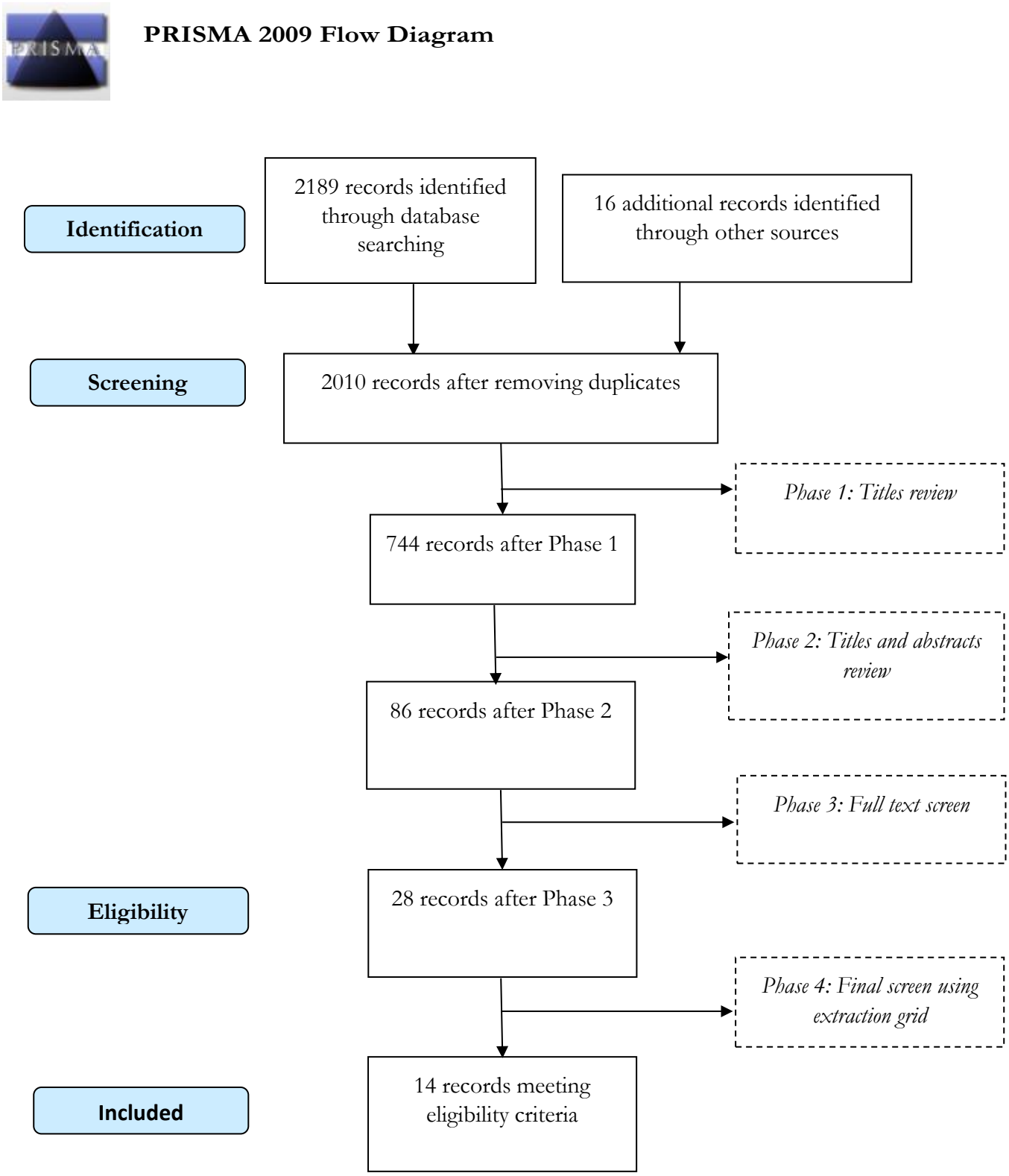
Phase 1: this involved a preliminary screening of titles to exclude studies that had ostensibly no relevance to the aims of the present review (e.g., they had no reference to TBI and/or adverse behavioural outcomes). Such process reduced the number of potentially relevant studies to 744.

Phase 2: this involved screening both titles and abstracts of all studies identified as potentially relevant following Phase 1. Such process reduced the number of potentially relevant studies to 86.

Phase 3: this involved full text screening of the remaining studies. Such process reduced the number of potentially relevant studies to 28.

Phase 4: this was the final stage of the study selection process. It involved examining whether each of the remaining studies met the inclusion/exclusion criteria described in 3.1 above. Such process was completed with the aid of an extraction grid developed using Microsoft Excel, summarising key information from each of the remaining studies against the eligibility criteria. Following this phase, 14 studies were considered suitable for the present systematic review.

Figure 1. Pictorial representation of the study selection process



4.2 Analysis of the studies

Table 1 and 2 provide with overviews of some of the key features of the final list of cross-sectional and longitudinal studies respectively, including year, country, assessment tools, population main characteristics and main findings. Studies were all published between 1998 and 2016, with twelve out of fourteen studies taking place in the last ten years. The findings of the systematic review will now be described.

4.2.1 Sample characteristics

4.2.1.1 Main features

The study populations of those who had experienced a paediatric TBI were made of offenders in five studies, patients in five studies, and individuals recruited from the community in two studies. In the two remaining studies, the study populations comprised a mixture of patients and individuals recruited from the community. It was not possible to compute the total number of participants across all studies because sample sizes for those who had had a paediatric TBI were not always reported. Overall, reported sample sizes of those who had sustained a paediatric TBI varied widely, from eleven (Dooley et al., 2008) to over 22,000 (Fazel et al., 2011).

Information regarding the number of male versus female participants who had sustained a TBI was only reported by eight studies, with the proportion of male participants ranging from 51.1% to 100%. Three studies comprised male participants exclusively, whereas in another three the proportion of male participants was over 70%. In a number of studies, details regarding gender ratio was only reported for the whole study population (including those with no history of TBI, or those who had TBI during adulthood; e.g., Fishbein et al., 2016). The studies were conducted across seven different countries, with the largest contributions from the US, UK and New Zealand (three studies each).

4.2.1.2 Overlap in participants

It is likely that there was a degree of overlap with respect to samples of participants tested by some longitudinal studies. This relates to the studies by Fishbein et al. (2016) and Brewer-Smyth et al. (2015), both examining an inmate population recruited from the same US institution between 2009 and 2010, and also the studies by Scott et al. (2014) and McKinlay et al. (2014), both examining clinical and non-clinical participants recruited in New Zealand. Such publications were included in the review despite overlapping cohorts as they used different although related aspects of antisocial behaviour (e.g., history of committing a violent crime versus self-reported aggression towards others; Brewer-Smyth et al., 2015; Fishbein et al., 2016). Thus, it was deemed that the studies would

still be valuable contributions to expand our understanding of the relationship between TBI and antisocial behaviour.

4.2.2 Methodological considerations

4.2.2.1 Design

Out of the final selection of fourteen studies, ten had a cross-sectional and four had a longitudinal design. Eight cross-sectional studies, and all longitudinal studies included a control group. In six cross-sectional studies and in two longitudinal studies, the control group comprised individuals who had never sustained a TBI (Andrews et al., 1998; Fazel et al., 2011; Fishbein et al., 2016; Dooley et al., 2008; Luukkainen et al., 2012; McKinlay et al., 2014; Timonen et al., 2002; Vaughn et al., 2014); in two cross-sectional and two longitudinal studies, the control group comprised individuals who had sustained an orthopaedic injury (i.e., that did not affect the skull or the brain, e.g., a limb fracture; McKinlay et al., 2014; Ong et al., 1998; Scott et al., 2014). One study was descriptive in nature and did not run statistical analyses on the variables of interest (Chitsabesan et al., 2015).

4.2.2.2 Temporal ordering of TBI and antisocial behaviour

Due to their nature of their design, cross-sectional studies present with limitations with respect to their ability to elucidate the temporal ordering of exposure and outcome variables, meaning that it may be difficult to ascertain whether TBI preceded the emergence of antisocial behaviour, or vice versa. The longitudinal studies included in the present review also varied in their ability to account for pre-injury evidence of antisocial behaviour. Ong et al. (1998) explored this by asking participants' parents to make retrospective ratings at the time of TBI of their children's pre-injury behavioural problems. McKinlay et al. (2014) examined parental reports of behavioural problems obtained from one to five years of age. In the studies by Timonen et al. (2002) and Fazel et al. (2011), antisocial behaviour prior to TBI was not recorded. This was because the outcome variable of interest (number of crimes committed) was extracted by national crime registers in Finland and Sweden respectively. In these countries crimes are only registered after people's 15th birthdays (age by which all participants had already had their TBI); for this reason, in those studies it was not possible to collect information regarding pre-injury convictions.

Significant variation across studies was also noted with respect to the amount of time passed between TBI and assessment of antisocial behaviour. Overall, mean age at TBI ranged up to sixteen years, and mean age at testing ranged from nine (Ong et al., 1998) to 51 (Fazel et al., 2011). Overall these methodological considerations all have implications for establishing the directionality

of the relationship between paediatric TBI and antisocial behaviour, which will be discussed in more detail in the Discussion section.

4.2.3 Assessment tools

4.2.3.1 Assessment of TBI

TBI was generally conceptualised as evidence of head trauma and altered consciousness. Studies varied however in terms of how history of this was established. Five studies relied on self-report, either through administration of previous semi-structured interviews (e.g., the Ohio State University TBI Identification tool, Corrigan & Bogner, 2007, used e.g. by Fishbein et al., 2016), or research-specific questions (e.g. asking participants "whether they had ever experienced a head injury, which caused unconsciousness or needed medical attention"; Vaughn et al., 2014). Nine studies relied on clinical diagnoses obtained through examination of participants' medical records.

Five cross-sectional studies (Andrews et al., 1998; Chitsabesan et al., 2015; Davies et al., 2012; McKinlay et al., 2014; Scott et al., 2014) and one longitudinal study (Ong et al., 1998) further divided participants into groups depending on the severity of their TBI. There was some variation with respect to how severity was defined; Andrews et al. (1998), McKinlay et al. (2014), and Scott et at al. (2014) relied on a combination of the findings from the Glasgow Coma Scale (GCS; Teasdale & Jennett, 1974), duration of loss of consciousness and post-traumatic amnesia, and radiology results; Chitsaseban et al. (2015) used the Rivermead Post-Concussion Symptoms Questionnaire (King et al., 1995), a measure of post-concussion symptoms; Davies et al. (2012) relied on length of loss of consciousness; Ong et al. (2014) relied on coma duration and GCS score. The longitudinal study by McKinlay et al. (2014) divided participants depending on whether they had been admitted to hospital following the TBI as outpatients or inpatients; such distinction might also reflect group differences in TBI severity.

4.2.3.2 Assessment of antisocial behaviour

As discussed in 3.1.1.2, antisocial behaviour is a heterogeneous concept; consistent with this, studies varied with respect to what types of antisocial activities they assessed, and the instruments that they used to assess them.

Ten studies relied on self-report. Among these, two required participants to complete previous self-report questionnaires (Andrews et al., 1998; Fishbein et al., 2016); one relied on parental report of aggression and delinquency (Ong et al., 1998), and one relied on a combination of previous measures completed by participants and their parents (Dooley et al., 2008). Remaining studies required participants to report on their history of violent and/or non-violent delinquency and

arrests (Brewer-Smyth et al., 2015; Davies et al., 2012; McKinlay et al., 2014; McKinlay et al., 2014; Scott et al., 2014; Vaughn et al. 2014).

Four studies examined participants' criminal case records, obtained through national registers. Among these, one examined history of violent offences (Chitsabesan at al., 2015); one examined history of both violent and non-violent offences (Luukkainen et al.; 2012); two examined history of criminal convictions overall (Fazel et al., 2011; Timonen et al., 2002).

4.2.4 Summary of the findings

4.2.4.1 Cross-sectional studies

Overall, all ten cross-sectional studies supported the presence of a link between paediatric TBI and engagement in antisocial activities. Participants with a history of TBI were consistently found to engage more frequently in antisocial activities than those without a history of this or with a history of orthopaedic injuries, regardless of type of population examined. For instance, in Vaughn et al. (2014) offenders who had a TBI showed higher levels of delinquency, bullying, and peer antisocial influence. Within clinical and community populations, TBI was found to be significantly associated with offending (Scott et al., 2014; McKinlay et al., 2014), with Luukkainen et al. (2012) showing a significant association between TBI and both violent and non-violent types of crimes. Aggression levels were also found to be higher in those who had experienced a TBI, both within a child patient population (Andrews et al., 1998), and in adult offenders (Fishbein et al., 2016).

It was also noted that history of TBI was markedly common among offenders. For instance, both Chitsaseban et al. (2015) and Davies et al. (2012) found that more than half of the inmates participating to their studies (64% and 72.1% respectively) suffered from a TBI at some point during childhood or adolescence.

4.2.4.2 Longitudinal studies

The four longitudinal studies included in the review also supported the presence of a link between TBI and engagement in antisocial behaviour, in all types of populations. For instance, Ong et al. (1998) found that children with history of TBI showed higher levels of aggression and delinquent behaviour according to their parents following the injury and compared to non-injured children. Timonen et al. (2012) and Fazel et al. (2011) found an increased risk for criminal offending in individuals during their lifetimes following a TBI. It should be noted however that in the study by Fazel et al. (2011) such risk was demonstrated for all people suffering from a TBI at some point their lives; no analyses were conducted for people who had their TBI in paediatric age. The study by McKinlay et al. (2014) further expanded on the findings, showing some subtle differences in

outcomes depending on age at TBI and whether participants had been admitted to hospital as outpatients versus inpatients.

4.2.4.3 Other relevant factors

A number of different factors were found to influence the association between paediatric TBI and antisocial behaviour, such as: number of, age at, and severity of TBI; drug and alcohol use; emotional and cognitive dysregulation; adverse life events; type of outcome measure used.

4.2.4.3.1 Number of TBIs

There was some tentative evidence linking higher number of TBIs with increased levels of antisocial behaviour. In Davies et al. (2012) there was a near significant contrast indicating that violent offending score (in terms of frequency and severity) was higher in those with more than four TBIs than in those with four or fewer TBIs. In Brewer-Smyth et al. (2015), within a population of young inmates, committing a violent crime was associated with a higher average number of TBIs by age fifteen compared to those who had not committed a violent crime; however, it should be noted that there was no difference in magnitude, as each group reported having suffered from one TBI on average.

4.2.4.3.2 Age at TBI

Some studies examined whether earlier age at TBI may be a risk factor for more adverse outcomes. Evidence for this was somewhat mixed. Fishbein et al. (2016) found that offenders who had their TBI before the age of thirteen had higher aggression scores than those who had their TBI later in life. In line with this, in McKinlay et al. (2014), age at injury was shown to be one of the strongest predictors of offending behaviour. Although these findings would appear suggestive of a link between earlier TBI and more adverse outcomes, the longitudinal study by Fazel et al. (2011) showed a different picture, as individuals who had their TBI before the age of sixteen were found to be at lower risk of criminal conviction than participants who had it later. A plausible explanation for this discrepancy in findings might relate to differences in health services provision across countries. The study by Fazel et al. was conducted in Sweden, where the justice system is known to have a particularly strong focus on rehabilitative approaches for young offenders (Hollander & Tärnfalk, 2007; Janson, 2004). Participants might have thus been more likely to receive rehabilitative interventions that prevented their behaviours from escalating further. If that was the case, this finding would further highlight the importance of early detection and treatment for at-risk individuals.

4.2.4.3.3 Severity of TBI

There was mixed evidence as to whether severity of antisocial behaviour varies as a function of severity of TBI. Andrews et al. (1998) found no significant difference in aggressive/antisocial behaviour between three groups of patients differing in injury severity (mild, moderate, severe). In contrast, Ong et al. (1998) found that patients with severe TBI showed significantly higher levels of delinquency and aggressiveness than both the moderate TBI group and control participants. Subsequently, Scott et al. (2014) found significant group differences in self-reported levels of criminal behaviour, with participants with moderate/severe TBI having the highest offending rates, followed by those with mild TBI, and finally those with orthopaedic injuries. It is worth noting that the study by Andrews et al. (1998) had relatively small sample sizes, and so might not have had sufficient statistical power to detect group differences; this might potentially help to explain discrepancies in the findings.

4.2.4.3.4 Drug and alcohol use

Two studies examined how substance use might intersect the relationship between paediatric TBI and antisocial behaviour. McKinlay et al. (2014) found that, when they accounted for history of alcohol and drug dependence during adolescence and early adulthood, there were no longer significant associations between TBI and offending for those injured up to five years of age. However, outcomes for those injured later in life remained substantially the same. Fishbein et al. (2016) found that, the lower the age that offenders started using drugs, the greater the total aggression score. Overall, these findings hint that drug and alcohol abuse in addition to paediatric TBI have an incremental effect in leading to adverse behavioural outcomes. It is possible that for those injured particularly early, alcohol and drug dependence have a stronger influence than TBI in determining whether someone is likely to engage in antisocial acts (McKinlay et al., 2014).

4.2.4.3.5 Emotional and cognitive dysregulation

Fishbein et al. (2016) examined whether emotional and cognitive dysregulation mediate the relationship between age at TBI (before versus at or after thirteen years of age) and aggression. It was then shown that when emotional dysregulation was added as a mediator, age at first TBI was no longer a significant predictor of aggression, for both age groups. There was no mediation effect for cognitive dysregulation for participants reporting a TBI at or after the age of thirteen. However, there was a partial mediation effect for those who had their TBI before thirteen years of age. Thus, although TBI before thirteen years of age still had a significant, direct effect on aggression, this was reduced by the indirect effect of cognitive dysregulation. These findings potentially indicate that emotional regulatory deficits may play a prominent role in explaining the association between

TBI and antisocial behaviour. Cognitive regulation deficits might be of more relevance for those injured earlier in life.

4.2.4.3.6 Environmental stressors and adverse life events and

Few studies controlled for environmental stressors in their designs, typically focusing on socio-economic status, parental criminality and parental substance use (Fazel et al., 2011; Luukkainen et al., 2012; McKinlay et al., 2014; Timonen et al., 2002; Vaughn et al., 2014); accounting for these factors did not alter the pattern of findings significantly, as TBI remained associated with antisocial behaviour. An exception was the study by Brewer-Smyth et al. (2015). They found that after accounting for several childhood adverse life events, such as sexual and emotional abuse, TBI before the age of fifteen years was negatively, rather than positively, associated with committing a violent crime.

4.2.4.3.7 Type of outcome measure used

One study (Dooley et al., 2009) provided some tentative evidence that different outcome measures may vary in their sensitivity at detecting delinquent and problem behaviours in children with history of TBI. In particular, theoretically-driven measures (i.e., based on a theoretical framework, such as e.g. the Social Learning Theory) were found to be more suitable than global psychopathology screening measures at detecting specific behaviour problems such as aggression in children with TBI.

4.2.5 Quality of evidence

The quality of the studies included in the review ranged from "poor" to "good". Four cross-sectional studies were classified as "good"; three were classified as "fair" and the remaining three were classified as "poor". All longitudinal studies were rated as "good", apart from one (Ong et al., 1998), which was classified as "fair". Common limitations across studies that impacted on their quality included: unclear temporal ordering of TBI and onset of antisocial behaviour; use of assessment tools of dubious validity/reliability; not accounting for other possibly important moderators or mediators in the analyses; relatively small sample sizes; lack of appropriate control groups; overlapping cohorts of participants with other studies.

Table 1. Cross-sectional studies examining the association between paediatric TBI and antisocial behaviour.

Authors	Year	Country	TBI population	Final TBI samples N (% Male)	TBI group mean age (SD)	Definition of TBI	Assessment of TBI	Assessment of antisocial behaviour	Main findings	Additional findings of note	Rating
D. Fishbein, J. K. Dariotis, P. L. Ferguson and E. E. Pickelsimer	2016	South Carolina (US)	Offenders	149 (N.R.) of total TBI sample had it before 13 years of age	N.R. for those who had a TBI before 13 years of age	Injury following from "a blow to the head or neck, resulting in an alteration of consciousness (i.e., dazed or confused, or forgetting what happened before or after the injury)"	Self-report (modified version of the OSU TBI-ID)	BPAQ (self-report questionnaire measuring aggression)	• Compared to the nTBI group, those with a TBI by age 13 reported higher total aggression scores (β = 8.65).	• Participants with a history of TBI before 13 years of age had higher aggression scores than those who had a TBI at or after that age • The lower the age at first drug use, the greater the total aggression score, regardless of whether TBI occurred before or after 13 years of age • No association between alcohol consumption during the 12 months prior to incarceration and total aggression in the TBI group • When emotional dysregulation is added as a mediator, estimates for age at first TBI are no longer significant predictors of aggression, and the indirect effect through emotional dysregulation is significant for both TBI age groups • No mediation effect for cognitive dysregulation • However, partial mediation effect for participants who had a TBI before 13 years old	*Good*
R.K. Brewer-Smyth, M. E. Cornelius and E. E. Pickelsimer	2015	South Carolina (US)	Offenders	N.R. for people who had a TBI before 15 years of age	N.R. for those who had a TBI before 15 years of age;	Injury following from "a blow to the head or neck, resulting in an alteration of consciousness (i.e., dazed or confused, or forgetting what happened before or after the injury)"	Self-report (modified version of the OSU TBI-ID)	Research-specific questions asking if participants had ever committed a violent crime in their lifetimes (e.g., physical or sexual assault)	• Committing a violent crime was associated with a higher average number of TBIs by age 15 compared to those who had not committed a violent crime (p=0.04) crime. • However, no significant difference in magnitude (essentially 1 TBI on average)	• When adjusting for childhood sexual abuse, childhood emotional abuse, childhood neighbour adversity, age, and gender, TBI by the age of 15 years was negatively associated with violent crime	*Fair*

M. G. Vaughn, C. P. Salas-Wright, M. Delisi and B. Perron	2014	US ("Pathway to Desistance"Study)	Offenders	411 (91.5%)	M=16.15 (1.14)	Head injury "which caused unconsciousness or needed medical attention"	Self-report (research-specific question)	Research-specific self-report measures of violent and non-violent delinquency, lifetime adolescent gang involvement and bullying behaviour, and peer delinquency and antisocial influence	• Compared to participants with no history of TBI, the TBI group reported higher levels of delinquency (t=5.41, p<0.001, d = 0. 32), bullying ($\chi 2$ = 10.07, p<0.01, φ = 0.09), peer delinquency (t=4.50, p<0.001, d = 0. 26) and peer antisocial influence (t=4.49, p<0.001, d=0.25)	-	*Poor*
P. Chitsabesan, C. Lennox, H. Williams, O. Tariq and J. Shaw	2015	UK ("CHAT" Study)	Offenders	93 (100%)	M=16.9 (0.7)	Head injury that "caused them to be knocked out or dazed"	Self-report (CHAT and RPQ questionnaire)	Case records of violent offences	• 64% of participants with history of TBI reported violent offences	-	*Poor*
R. C. Davies, W. H. Williams, D. Hinder, C. N. W. Burgess and L. T. A. Mounce	2012	UK	Offenders	61 (100%)	M=16.87 (N.R.)	Head injury that "caused [them] to be knocked out and/or dazed and confused for a time."	Self-report (research-specific question)	Research-specific structured interview assessing history of violent offending	• 72.1% participants reported suffering a TBI at some point in their lives	• No significant main effect of either frequency or severity of TBI on violent offending • Near significant contrast indicating that violent offending score (frequency & severity) was higher in those with more than 4 TBIs than in those with 4 or fewer TBIs • People with no history of TBI or mild concussions reported the age of their first conviction as significantly older than those who had experienced moderate/severe TBI	*Fair*
S. Luukkainen, K. Riala, M. Laukkanen, H. Hakko and P. Rasanen	2012	Finland ("STUDY-70" Project)	Clinical (psychiatric inpatients)	26 (69.2%)	M=15.2 (1.2)	Diagnosis of either "fracture of skull bones (excluding facial traumas), intracranial injury or injury of cranial nerves according to the ICD-9 or ICD-10 diagnostic criteria" and requiring hospital admission	Clinical records	Criminal records	• TBI was associated with criminality (p<.001). • Participants with a history of TBI had committed more violent (p<.001) and non-violent crimes (p<.001) than those without a history of TBI	• After adjusting for gender, age and family type of adolescents, parents' employment status, compared to the no TBI group, in the TBI group the likelihood of committing any crime was 4.9-fold; the likelihood of committing violent crimes was 5.9-fold, and for non-violent crimes it was 3.9-fold	*Good*

J. J. Dooley, V. Anderson, S. A. Hemphill and J. Ohan	2008	Australia	Clinical	11 (100%)	M=15.7 (1.3)	Moderate TBI: "GCS scores of 9–12, LOC of 1–24 hours and/or radiological evidence of brain abnormalities" Severe TBI: "lowest GCS scores of 3–8, LOC>24 hours and/or radiological evidence of brain abnormalities"	Clinical records	• Aggression and delinquent behaviour sub-scales of the CBCL (Parent-completed questionnaire) • YSR (self-report questionnaire of aggressive and delinquent behaviours) • RPQ-YR (self-report questionnaire of aggression) • RPQ-IR (parent-completed questionnaire of aggression) • FAS (self-report questionnaire of aggressive behaviour)	• On the YSR, no difference between children with versus without a history of TBI in aggression (χ=1.54, p=.12) or delinquent behaviour (χ=1.20, p=.23). • On the CBCL, no group difference in aggression (χ=.11, p=.91) or delinquent behaviours (χ=.95, p=.34). • On the RPQ-YR, the TBI group reported more reactive (χ=2.54, p=.01), proactive (χ=2.99, p=.003) and total aggression (χ=2.77, p=.006). • On the RPQ-IR, no group difference in reactive (χ=.51, p=.61), proactive (χ=.56, p=.58) or total aggression (χ=.50, p=.62). • The TBI group however reported more reactive overt and pure overt aggression (χ=2.75, p=.006) and engaged in more pure overt aggression (χ=2.98, p=.003). • On the FAS, no group differences in instrumentally aggressive behaviours (χ=.42, p=.67).	• Global measures of psychopathology (e.g., YSR) are less sensitive at detecting differences in aggressive behaviours between injury groups than theoretically-driven measures (e.g., RPQ)	*Good*

T. K. Andrews, F. D. Rose and D. A. Johnson	1998	UK	Clinical	10 with severe TBI (N.R.) 9 with moderate TBI (N.R.) 8 with mild TBI (N.R.)	Severe: M= 12.7 (3.2) Moderate TBI: M=12.0 (2.9) Mild TBI: M=12.5 (3.0)	Diagnosis of TBI "according to the ICD-9 criteria (codes 800± 804, 850± 854)"	Clinical records	DAABS (assessing aggressiveness and antisocial behaviour)	• Children with history of TBI showed higher levels of aggressive/antisocial behaviour than those with no history of TBI (t=-19.3, p <0.05)	• No significant difference among TBI groups in terms of aggressive/antisocial behaviour	*Poor*
C. Scott, A. McKinlay, T. McLellan, E. Britt, R. Grace and M. MacFarlane	2014	New Zealand	Mixed (clinical and community)	65 (66.2%) with moderate/severe TBI 61 (54.1%) with mild TBI	Moderate/severe TBI: M=23.15 (3.52) Mild TBI: M=22.31 (2.78)	Moderate TBI: GCS of 9 –12 or higher if accompanied by radiological abnormalities, PTA less than one week, and LOC less than 6 hrs. Severe TBI: PTA greater than 1 week, or LOC for greater than 6 hrs or GCS less than 9	Clinical records	Research-specific structured interview (assessing e.g., history of assault, drunk and disorderly behaviour, vandalism)	• Significant group difference in offending (p<.001), with participants with moderate/severe TBI having the highest rates, followed by those with mild TBI, and finally those with orthopaedic injuries.	-	*Fair*
A. McKinlay, R. C. Grace, T. McLellan, D. Roger, J. Clarbour and M. R. MacFarlane	2014	New Zealand	Mixed (clinical and community)	62 with moderate/severe TBI (66.1%) 62 with mild TBI (53.2%)	Moderate/severe TBI: M= 23.29 (3.55) Mild TBI: M = 22.31 (2.78)	Moderate/severe TBI: "clinical diagnosis of moderate or severe TBI, skull fracture or evidence of lesion on computed tomography; cerebral hemorrhage or PTA of more than 24 hours." Mild TBI: a "clinical diagnosis of mild TBI; LOC of less than 20 minutes; PTA of less than 1 hour; no evidence of skull fracture or lesion on computed tomography"	Clinical records	Research-specific structured interview (assessing e.g., history of assault, drunk and disorderly behaviour, vandalism)	• Compared to people with orthopaedic injuries, the moderate/severe TBI group was more likely to have a history of offending behaviour (OR=20.35, p<.01) and conviction (OR=8.88, p<.05), to have been arrested (OR=12.07, p<.05), fined (OR=6.0, p<.01), to have committed motor vehicle accident offences (OR=6.0, p<.01), and/or to have a history of petty crime (OR=8.88, p<.05). • The mild TBI group was at increased risk of history of offending (OR=8.66, p<.05)	• The strongest predictors of offending behaviour were TBI status, higher levels of malevolent aggression, and age at injury	*Good*

Note: N.R.=not reported; OSU TBI-ID= Ohio State University TBI Identification tool (Corrigan & Bogner, 2007); BPAQ=Buss Perry Aggression Questionnaire (Diamond, Wang, & Buffington-Vollum, 2005); CHAT =Comprehensive Health Assessment Tool Study (Chitsabesan et al., 2014); RPQ= Rivermead Post-Concussion Symptoms questionnaire (King et al., 1995); ICD-

9=International Classification of Diseases, Ninth Revision; ICD-10=International Classification of Diseases, Tenth Revision; GCS=Glasgow Coma Scale (Teasdale & Jennett, 1974); LOC=loss of consciousness; CBCL=Child Behaviour Checklist (Achenbach, 1991); YSR=Youth Self-Report (Achenbach, 1991); RPQ-YR=Reactive–Proactive Questionnaire–Youth Report (Raine et al., 2006); RPQ-IR=Reactive–Proactive Questionnaire–Informant Report (Brown et al., 1996); FAS= Form of Aggression Scale (Little et al., 2003); DAABS= Aggressive and Antisocial Behaviour Scales (DeBlois & Stewart, 1988); PTA=Post-Traumatic Amnesia.

Table 2. Longitudinal studies examining the association between paediatric TBI and antisocial behaviour.

Authors	Year	Country	Population	Cohort/Time points	Final Samples N (% Male)	Definition of TBI	Assessment of TBI	Assessment of antisocial behaviour	Main findings	Additional findings of note (e.g. moderators/mediators)	Rating
A. McKinlay, J. Corrigan, L. J. Horwood and D. M. Fergusson	2014	New Zealand (the "Christchurch Health and Development Study")	Clinical	• Participants assigned to subgroups depending on age of TBI (0-5, 6-15, 16-21) • For those who had TBI before 0-15 years of age, antisocial behaviour was examined over 16 to 25 years of age	TBI between 0-5 years of age: • 55 (N.R.) outpatients • 22 (N.R.) inpatients TBI between 6-15 years of age: • 55 (N.R.) outpatients • 35 (N.R.) inpatients	Moderate/severe TBI: "clinical diagnosis of moderate or severe TBI", skull fracture or evidence of lesion on computed tomography; cerebral hemorrhage or PTA of more than 24 hours." Mild TBI: "clinical diagnosis of mild TBI; LOC of less than 20 minutes; PTA of less than 1 hour; no evidence of skull fracture or lesion on computed tomography."	Parental interview, parental diaries, and medical records	• SRDI (self-report questionnaire, assessing e.g. property and violent offences in the last 12 months) • Number of arrests	TBI between 0-5 years of age: (co-variates: gender, family SES at birth, and parental reports of behavioural problems from 1-5 years of age): • The inpatient group was at higher risk of arrests (IRR=4.33, p<.01) and property offenses (IRR=2.24, p<.01). • Both the outpatient and inpatient groups were more likely than those with no TBI history to have been involved in violent offences (IRR=1.47, p<.05; IRR=2.72, p<.01, respectively). TBI between 6-15 years of age: • The outpatient group was more likely to have been involved in property offences (IRR=1.44, p<.05), but not violence offences (IRR=0.98, p>.05) • The inpatient group was less likely to have been involved in a property offence (IRR=0.63, p<.05), but more likely to have been involved in a violent offence (IRR=1.50, p<.05)	Additional co-variates: alcohol dependence and drug dependence TBI between 6-15 years of age: No significant associations remained for any outcome of interest. TBI between 6-15 years of age: • The inpatient group remained significantly more likely to have committed violent offenses • The outpatient group was significantly less likely to have committed violent offenses • The other associations were no longer significant.	*Good*
L. C. Ong, V. Chandran, S. Zasmani and M. S. Lye	1998	Malaysia	Clinical	• Pre-injury scores taken retrospectively in acute phase of TBI • Post-injury scores taken 6 months after TBI • Mean age at TBI:	28 with severe TBI (N.R.) 28 with mild-to-moderate TBI (N.R.)	Severe TBI: "head injury", "GCS score <9, coma duration of 24+ hours" Mild-to-Moderate TBI: "head injury", "GCS score <9, coma duration ranging from a few minutes to six hours"	Clinical diagnosis	Aggression and delinquent behaviour scales of the CBCL (Parent-completed questionnaire)	• Group effect for delinquency (F=4.32, p=.002) and aggressiveness (F=3.69, p=.007), explained by significant differences between the severe TBI group and both the moderate TBI group and orthopaedic controls. • No pre/post-injury differences in the behaviour domains between the moderate TBI and orthopaedic group.	• The severe TBI group had significantly lower delinquency and aggression pre-injury scores than orthopaedic controls.	*Fair*

				Severe TBI group: M=9.8 (SD=1.68) Mild-to-moderate TBI: M=9.8 (SD=1.79)							
S. Fazel, P. Lichtenstein, M. Grann N. Långström	2011	Sweden	Community	• Population of individuals born between 1958 and 1994, followed-up until 2009 • TBI occurred up until 16 years of age • Antisocial behaviour assessed after 16th until end of follow-up	22,914 (71.1%)	Diagnosis of TBI "according to the ICD-8 (diagnostic codes 851–852), ICD-9 (codes 851–854), or ICD-10 (codes S06.01–S06.09) criteria"	Clinical diagnosis	Criminal records	• Participants were divided into 2 groups: TBI before versus after 16 years of age • Participants with TBI before 16 years of age were at lower risk of criminal convictions than those who had it after 16 years of age (x^2= 35.7, p=.001)	• After adjusting for age, gender, ad socio-demographic confounders, and substance abuse, participants with history of TBI were at increased risk of criminal convictions compared to people with no history of TBI (NB: in this analysis TBI might have occurred at any age, not only before 16 years of age)	*Good*
M. Timonen, J. Miettunen, H. Hakko, P. Zitting, J. Veijola, L. von Wendt and P. Rasanen	2002	Finland (the "Northern Finland 1966 Birth Cohort Study")	Community	• TBI might have occurred up until fifteen years of age • Antisocial behaviour followed up from fifteen until thirty-one years of age	10934 (51.1%)	Diagnosis of "skull fracture, cerebral contusion and concussion and intracranial injuries sustained as a result of trauma, using the ICD-9 diagnostic criteria (codes 800–801, 803, 804 except for facial traumas, 850–854 and 950–951)."	Clinical diagnosis	Criminal records	• In male participants, TBI significantly increased the risk of criminal offending both before (OR 1.7, 1.1–2.6) and after (OR 1.6, 1.0–2.5) adjusting for marital status of the mother and social class of the father at the time of birth, dwelling place in 1980 (urban/rural) • In female participants with TBI, the risk of criminality was OR 1.5, 0.4–6.1)	• Criminals who sustained the TBI before the age of 12 started their criminal career significantly earlier compared to those who had the TBI after the age of 12	*Good*

Note: SRDI=Self-Report Delinquency Inventory (Elliott & Huizinga, 1989); CBCL=Child Behaviour Checklist (Achenbach, 1991).

Discussion

5.1 Summary of the findings

Despite growing evidence in the literature supporting an association between paediatric TBI and antisocial behaviour, a paucity of work has examined this systematically. The present review conducted a systematic search of four main databases, with the aims of increasing current understanding of whether paediatric TBI is a likely risk factor for later engagement in antisocial acts, and what other biopsychosocial factors might intersect the relationship between these two variables. A total of fourteen articles meeting eligibility criteria were identified; notably, twelve out of these were published within the last 10 years, denoting a recent increased interest in the area. Antisocial behaviour is currently considered a burgeoning issue at both individual and community level (Great Britain Home Office, 2004); it is therefore unsurprising that more time and effort are currently being invested towards the study of its risk factors. The current review appears timely in reviewing current progress in the area.

Taken together, the studies included in the current review confirm the presence of a significant association between paediatric TBI and antisocial behaviour. Overall, people who experienced a TBI before the age of nineteen were found to be at higher risk of aggression, arrests, offences, and violence. The studies also offered some tentative, preliminary evidence that the relationship between paediatric TBI and antisocial behaviour is not linear, and instead is likely to be influenced by a number of different variables. There was, for example, tentative evidence that levels of antisocial behaviour vary as a function of number and severity of TBI and age at injury (e.g., before and after 16). Difficulties around emotion regulation might play a key mediating factor in explaining the relationship between TBI and aggression, and to a greater extent than cognitive regulation deficits, although these may still be relevant for those injured earlier in life. It was hinted that for people injured earlier on in their lives, drug and alcohol use might be a more prominent risk factor for criminal behaviour than TBI, and that the earlier people start using drugs, the more severe their behavioural problems. Adverse life events during childhood such as history of abuse also appear to be factors of interest; however since these have been included as confounder variables, their role in influencing the nexus between TBI and antisocial behaviour is relatively unclear.

5.2 Limitations of current evidence

The studies overall provided with some insight into the potential role of TBI and other biopsychosocial variables in influencing later engagement in antisocial acts. However, the review included only fourteen studies. Based on their findings alone, it is difficult to draw definite

conclusions regarding both the nature of the relationship between paediatric TBI and antisocial behaviour, and how and to what extent different variables intersect this association. It is also worth noting that most studies were characterised by several consistent limitations, which further impact on our ability to make definite claims about the findings. Firstly, a major limitation is that the temporal ordering of TBI, onset of antisocial behaviour and of other critical variables (such as e.g. emotional and cognitive dysregulation) was not always established; in some cases it was not reported whether TBI occurred prior to, during or after offending. Excluding some exceptions, information regarding participants' pre-injury characteristics (e.g., pre-injury behavioural problems, neurocognitive functioning, social background) was often limited. Moreover, a number of studies did not include control groups, and data were not always evaluated statistically. It cannot be excluded that TBI and antisocial behaviour stem from other, shared biopsychosocial risk factors (e.g., risk-taking behaviours, low IQ, disadvantageous social backgrounds). It is also still possible that TBI is the manifestation (rather than the cause) of pre-existing behavioural problems, which in turn make children more prone to accidents and injuries (eventually leading to TBI).

Secondly, there was a marked degree of heterogeneity across studies in terms of design and methodology. Both TBI and antisocial behaviour were measured using a variety of different tools (e.g., semi-structured interviews, parental diaries, medical and criminal records); moreover, assessments were not always standardised. This limits our ability to make an appropriate comparison across studies, and raises questions regarding the validity and reliability of the findings. Several studies relied on self-report assessments of both TBI and antisocial behaviour; there is previous evidence that people can often fail to remember previous history of TBI, especially when this occurred at early age, leading to inaccuracies in their responses (McKinlay et al., 2016). History of antisocial behaviour may also tend to be under-reported due to social desirability and impression management biases (Edens, 2004).

Moreover, for most studies, sample comprised predominantly or even exclusively male participants. Some studies also involved overlapping cohorts. These limitations affect negatively the generalisability of the findings to the wider population. The time passed between TBI and assessment of antisocial behaviour ranged widely across studies, from a few months to several years. This makes it difficult to elucidate how TBI might interfere with developmental processes related to specific stages, and the long-term effects of TBI on adult functioning. Finally, TBI was defined and assessed in several different ways; this is recognised a major challenge for completing evaluations of previous literature in the field (Haydel, 2016). The review identified fourteen studies across seven different countries, varying in terms of policies, criteria for identifying and managing

antisocial behaviour, and practises. Although such heterogeneity is inevitable, it has implications for combining and interpreting the findings from different studies.

5.3 Recommendations and future directions

It is important that future investigations build on the aforementioned methodological flaws. The small pool of relevant literature indicates that this is an area deserving further attention, preferably employing sounder research designs that minimise the risk of biases and facilitate a more accurate evaluation of the directionality of such relationship. Recommendations for future research might include increased reliance on a combination of valid and reliable methodologies and sources for collecting more detailed information regarding history of TBI and antisocial behaviour; more detailed examination and inclusion of participants' pre-injury backgrounds, and other important psychosocial variables (e.g., neurocognitive functioning), to expand on the available evidence; use of control groups; recruitment of larger and more representative samples of participants.

The nature of the mechanisms through which TBI may lead to engagement in antisocial behaviour is also relatively unclear to date. Previous studies have hypothesised that such link might be at least partly mediated by damage to the frontal part of the brain following a TBI. The brain frontal lobes are thought to be the most common region of injury caused by TBI, possibly due to their positioning at the front of the cranium, large size and proximity to the sphenoid wing (Levin et al., 1987). They are responsible for numerous functions associated with successful everyday social functioning, such as for instance impulse control, impulsivity, inhibition, planning and problem-solving; impairments in these cognitive domains have all previously linked to antisocial behaviour (see e.g., Ogilvie et al., 2011). Yet, to our knowledge there is limited work formally testing the mediating role of such neuropsychological impairments. It is also worth noting that most previous studies have focused on the examination of relatively limited numbers of biopsychosocial variables that might affect the link between TBI and antisocial behaviour. There is robust evidence within the field of developmental psychology that the study of the effect of isolated risk factors is unlikely to advance significantly our understanding of the antecedents of problem behaviours. Instead, it has been increasingly shown that risk factors are better examined in combination, and that their cumulative effect should be considered (e.g., Rutter, 1979). Biopsychosocial risk factors should ideally cover both child- and environmentally-based stressors, such as parenting, since both types have been implicated in the development of behavioural issues (see e.g., Dodge & Pettit, 2003).

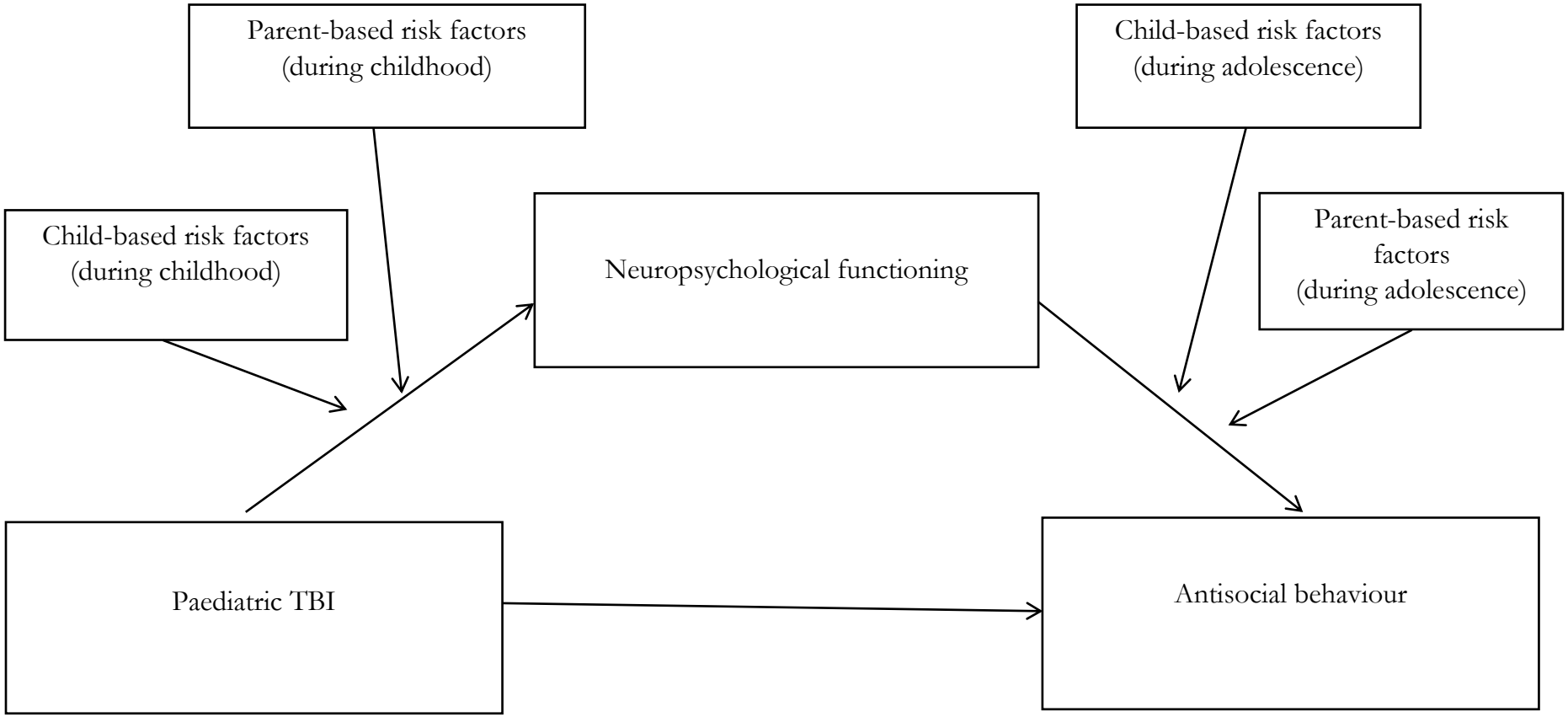
The next chapter of the present thesis aims to expand on these gaps in the literature and make a step forward developing a more holistic understanding of the link between paediatric TBI and antisocial behaviour. A novel, age-graded theoretical framework will be developed, integrating a

range of different risk factors inherent to both children's characteristics (e.g., early temperament) and environment (e.g., family adversity) during development and adolescence. The possible mediating role of neuropsychological impairments following TBI will also be examined (see Figure 2 below for an outline of the model that will be examined in the next chapter). This will aim to provide a more comprehensive coverage of the possible mechanisms explaining the link between paediatric TBI and antisocial behaviour, and individual and cumulative effect of different critical child- and parent-related variables during development. The model will be tested using data collected by the Avon Longitudinal Study of Parents and Children (ALSPAC), a large UK population-based study (Boyd et al., 2013). The study will also aim to address some of the methodological limitations of previous literature, such as e.g. unclear temporal ordering of TBI and antisocial behaviour, or use of cross-sectional designs.

5.4 Clinical implications

Notwithstanding its limitations, the findings of the present review have potentially important implications for justice and rehabilitation systems. Concerns have been raised regarding limited understanding both within the general public and professionals regarding the possible effects of brain injuries on overall functioning (Hawley, 2003; McKinlay, 2014). There are currently significant percentages of young people living in custody within secure settings in the UK who might have undiagnosed and/or untreated neurological issues such as TBI; although the presence of such conditions might have contributed to the behaviours that led them to offend, it is rare that history of brain injury is assessed or considered by criminal justice professionals in evaluating offenders' rehabilitative needs (Hughes et al., 2012; Williams, 2012). The findings of the present review corroborate the presence of an association between paediatric TBI and antisocial behaviour, and therefore further confirm the importance of information provision to promote early identification and treatment. They highlight the need for criminal and health system to be aware of this link to ensure that sufferers of TBI can access appropriate medical and rehabilitation services in a timely manner. This may be pivotal importance in reducing burdening impact and costs linked with antisocial behaviour at individual, societal and financial level.

Figure 2. Pictorial representation of the theoretical model that will be tested in the next chapter, examining the association between paediatric TBI and engagement in antisocial behaviour, and the intersecting role of various child- and parent-based risk factors.



5.5 Conclusions

In summary, the present review evaluated systematically previous literature examining the association between paediatric TBI and later engagement in antisocial behaviour. Taken together, the findings provide tentative support for the presence of a relationship between these variables. Some potentially intersecting factors (e.g., drug and alcohol use) were also identified. Overall, it was noted that only a small pool of literature has been dedicated to this topic to date, and that this is characterised by several methodological flaws. These limitations affect our ability to draw definite conclusions from the findings, and highlight the need for more investigations using sounder research designs. The present findings contributed to enhance our understanding of current progress in the area and have implications for informing preventative and rehabilitative measures.

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Appendix A: Search strategies

Ovid MEDLINE, Embase and PsycInfo

1. "traumatic head injur*".mp. [mp=ti, ab, hw, tn, ot, dm, mf, dv, kw, fs, nm, kf, px, rx, ui, sy, tc, id, tm]
2. "traumatic brain injur*".mp. [mp=ti, ab, hw, tn, ot, dm, mf, dv, kw, fs, nm, kf, px, rx, ui, sy, tc, id, tm]
3. exp Brain Injuries/
4. exp Traumatic Brain Injuries/
5. tbi.mp.
6. "acquired brain injur*".mp.
7. ABI.mp.
8. "intracranial injur*".mp. [mp=ti, ab, hw, tn, ot, dm, mf, dv, kw, fs, nm, kf, px, rx, ui, sy, tc, id, tm]
9. exp Child Behaviour Disorders/
10. Anti?social behavio?r.mp.
11. exp Juvenile Delinquency/
12. exp Conduct Disorder/
13. conduct.mp.
14. exp Violence/
15. violen*.mp.
16. exp "Disruptive, Impulse Control, and Conduct Disorders"/
17. exp Aggression/
18. aggress*.mp.
19. "disrupt* behavio?r*".mp.
20. exp "Attention Deficit and Disruptive Behaviour Disorders"/
21. exp Problem Behaviour/
22. exp Crime/
23. exp Criminal Behaviour/
24. crime*.mp.
25. criminal*.mp.
26. exp Child Psychiatry/
27. exp Young Adult/
28. exp Psychology, Child/
29. exp Adolescent/
30. child*.mp.
31. adolescen*.mp.
32. exp Child/
33. p?ediatric.mp.
34. exp Pediatrics/
35. teen*.mp.
36. juvenile.mp.
37. youth.mp.
38. "young people".mp.
39. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8
40. 9 or 10 or 11 or 12 or 13 or 14 or 15 or 16 or 17 or 18 or 19 or 20 or 21 or 22 or 23 or 24 or 25
41. 26 or 27 or 28 or 29 or 30 or 31 or 32 or 33 or 34 or 35 or 36 or 37 or 38
42. 39 and 41
43. 40 and 42

44. limit 43 to (english language and full text and humans and yr="1990 -Current" and journal article)
45. remove duplicates from 44

Web of Science

#30	#28 AND #17 Refined by: **PUBLICATION YEARS:** (2014 OR 2011 OR 2000 OR 2017 OR 2015 OR 2008 OR 1998 OR 1999 OR 2016 OR 2004 OR 2002 OR 1994 OR 2012 OR 2006 OR 1997 OR 1992 OR 2013 OR 2005 OR 2001 OR 1991 OR 2010 OR 2007 OR 1995 OR 1993 OR 2009 OR 2003 OR 1996)
#29	#28 AND #17
#28	#27 AND #7
#27	#26 OR #25 OR #24 OR #23 OR #22 OR #21 OR #20 OR #19 OR #18
#26	**TOPIC:** ("young adult*")
#25	**TOPIC:** ("young people")
#24	**TOPIC:** (youth*)
#23	**TOPIC:** (youth)
#22	**TOPIC:** (juvenile)
#21	**TOPIC:** (teen*)
#20	**TOPIC:** (p$ediatric)
#19	**TOPIC:** (adolescen*)
#18	**TOPIC:** (child*)
#17	#16 OR #15 OR #14 OR #13 OR #12 OR #11 OR #10 OR #9 OR #8
#16	**TOPIC:** (disrupt*)
#15	**TOPIC:** (delinquen*)
#14	**TOPIC:** (criminal)
#13	**TOPIC:** (crime)
#12	**TOPIC:** ("disrupt* behavio$r")
#11	**TOPIC:** (aggress*)
#10	**TOPIC:** (violen*)
#9	**TOPIC:** (conduct)
#8	**TOPIC:** ("anti$social behavio$r")
#7	#6 OR #5 OR #4 OR #3 OR #2 OR #1
#6	**TOPIC:** (intracranial injur*)
#5	**TOPIC:** (abi)
#4	**TOPIC:** (acquired brain injur*)
#3	**TOPIC:** (tbi)
#2	**TOPIC:** ("traumatic head injur*")
#1	**TOPIC:** ("traumatic brain injur*")

Empirical Research Project

The association between paediatric traumatic brain injury and antisocial behaviour in adulthood: a longitudinal study using the ALSPAC data

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1. Abstract

Previous literature suggests that paediatric traumatic brain injury (TBI) and antisocial behaviour are associated; yet the role of different child- and environmental biopsychosocial social factors in influencing this relationship is still not well-understood. Using the data collected by the Avon Longitudinal Study of Parents and Children (ALSPAC), the present study aimed to explore the association between TBI before the age of twelve, antisocial behaviour at the age of twenty, and a number of key child- and environmentally-related risk factors that have been previously associated with both TBI and problem behaviours. The findings revealed that parent-reported ADHD symptoms such as poor attention and inhibition, and self-reported substance use in adolescence mediated the association between paediatric TBI and antisocial behaviour; the presence of early emotional and behavioural difficulties moderated the link between TBI and attention and inhibition symptoms, whereby only those with such early emotional and behavioural difficulties were at risk of adverse outcomes later on in life. Such moderated mediation effect remained significant when comparing outcomes for people with TBI versus those with history of orthopaedic injury, whereas it became non-significant when comparing people with orthopaedic injury versus those with no history of any injury; these findings increase our confidence that the link between TBI and antisocial behaviour is causal in nature. The present research expands on previous literature by providing with a novel, time-graded model that offers a more holistic characterisation of the relationship between paediatric TBI, its long-term outcomes, and contribution of key individual and environmental variables. The implications of the findings with respect to informing preventative and rehabilitative interventions are discussed.

2. Introduction

Both traumatic brain injury (TBI) and antisocial behaviour represent key topics of public concern, with TBI being the leading cause of death and disability worldwide (Dinsmore, 2013), and management of antisocial behaviour costing over three billions a year to government agencies in the UK, according to recent estimates (Great Britain Home Office, 2004). Empirical support for the existence of an association between paediatric TBI and later engagement in antisocial and criminal activities was initially originated by a number of studies showing remarkably high rates of TBI within forensic populations, with these being as high as 70% in some cases (see e.g., Leon-Carrion & Ramos, 2003; Lewis et al., 1986, 1988; also see Allely et al. 2015 for a recent review). There is also evidence that previous history of TBI tends to be a risk factor for committing violent (versus non-violent) crime among prisoners (Leon-Carrion & Ramos, 2003). Youths with history of paediatric TBI also report earlier onset of criminal behaviour, and are at higher risk for reoffending compared to their uninjured counterparts (Perron & Howard, 2008).

One main limitation of studies with forensic populations is that it is unclear to what extent their findings and implications also apply to the community population (Parsonage, 2016). For instance, both TBI and offending may have other common underlying determinants, such as for instance socio-economic disadvantage (e.g., homelessness; Bremner et al., 2005; Kushel et al., 2005) and tendency towards engaging in risk-taking behaviours (Williams, 2013). However, as also reviewed in the Systematic Literature Review component of the present thesis, only a paucity of studies has been conducted to date with non-forensic communities; moreover, such studies have typically used cross-sectional designs, making it difficult to establish the temporal ordering and aetiology of the relationship between TBI and onset of antisocial behaviour (Bellesi et al., submitted). My systematic review found only four studies that had a longitudinal design (Fazel et al., 2011; McKinlay et al., 2014; Ong et al., 1998; Timonen et al., 2001). Although all supported an association between TBI before age of sixteen and engagement in antisocial activities (e.g., offences, arrests), only three had a large-scale design, and none had been conducted in the United Kingdom (UK). One of the studies compared outcomes for those with history of TBI before versus after age of sixteen, but not for those with a paediatric TBI versus uninjured control participants, meaning that the overall effect of paediatric TBI could not be established (Fazel et al., 2011). Only one study included a negative exposure control group comprising individuals with orthopaedic injury (Ong et al., 1998); lack of a negative exposure control group makes it harder to establish whether the adverse outcomes associated with TBI are likely to be due to suffering from any injury in general rather than TBI specifically. Finally, and only two studies out of the four

identified had been conducted in the last ten years. Since several advances have been made in the last decade with respect to both identification and rehabilitation of TBI (Dang et al., 2017), it is essential that the evidence is up to date to draw appropriate implications.

Notably, a study just recently published by Kennedy et al. (2017) overcame some of the limitations of previous literature by examining outcomes for a large sample of participants recruited from the Avon Longitudinal Study of Parents and Children (ALSPAC), a cohort study of children born in the former county of Avon, England, between 1991 and 1992 (Boyd et al., 2013; Fraser et al., 2013). Participants were divided into three groups, depending on whether they had suffered from a TBI or an orthopaedic injury before the age of sixteen, or had no history of any type of injury. Compared to the no injury group, both the TBI and orthopaedic injury groups were found to be at higher risk of offending, being in trouble with the police, and presenting with parent-reported conduct problems at the age of seventeen (Kennedy et al. 2017). Although the study by Kennedy and colleagues represents a solid step forward towards generating more methodologically robust evidence supporting the association between paediatric TBI and antisocial behaviour within a UK population, there are still several questions that remain unanswered. The study examined outcomes at the age of seventeen, meaning that longer-term outcomes in adulthood remain to be clarified. There is growing evidence that recovery from brain injury in paediatric age typically includes an immediate stage, characterized by rapid, dramatic gains in functional recovery, and a subsequent "latent" phase, which can last several years up until young adulthood. In this, young people tend to be subjected to more complex demands in their daily functioning, which, in turn, tend to clarify and make the "actual" impact of the brain injury on motor, cognitive and social performance more discernible (the "neurocognitive stall"; Chapman, 2006). This evidence indicates the importance of and need for more research investigating the long-term impact of TBI on subsequent development and functioning in adulthood (Bramlett & Dietrich, 2015).

Why might TBI lead to increased risk of antisocial behaviour?

The mechanisms through which paediatric TBI might lead to more problem behaviours are also still poorly understood to date. There is evidence that optimal brain development during childhood is uniquely dependent on the integrity of particular cerebral structures. Thus, if a cerebral region is damaged at a critical stage of development, cognitive skills dependent on that region may become irreversibly impaired (Kolb, 1995; Luciana, 2003). The brain frontal lobes are the most commonly damaged region by TBI, possibly due to their positioning at the front of the cranium, large size and proximity to the sphenoid wing (Levin et al., 1987). They are responsible for numerous functions involved in emotional and behavioural regulation, such as impulse control, impulsivity,

inhibition, planning and problem-solving; these are typically clustered under the umbrella term of "executive functions", and are pivotal for positive everyday social functioning (Anderson, 2002). The frontal lobes are among the last areas of the brain to fully mature and are typically not fully developed until early adulthood (see e.g., Anderson et al., 2001; Kelly, 2000; Klenberg et al., 2001). Damage to the frontal lobe in paediatric age might lead to complex neural pathways becoming prematurely disconnected, and therefore to long-term negative impact on the abilities to regulate everyday thinking and behaviours. In particular, there is evidence of a strong link between paediatric TBI and difficulties in executive skills such as attention (see e.g., Catroppa et al., 1999; Whyte et al., 1996) and impulse control (see e.g., Levin et al., 2004; Sinopoli et al., 2011). Difficulties with executive functions typically underpin attention-deficit/hyperactivity disorder (ADHD; see e.g., Willcutt et al., 2005); indeed, persistent patterns of inattentive and impulsive behaviour are main diagnostic criteria for diagnosis (American Psychiatric Association, 2013). On the basis of this, it is thus perhaps unsurprising that children with TBI are approximately three times more likely than their uninjured counterparts to receive a diagnosis of secondary ADHD, i.e. ADHD in the absence of pre-injury symptoms (Li & Liu, 2013).

Importantly, difficulties around inattention and impulsivity are known to increase the likelihood that people engage in risk-taking and reckless activities positing a risk to the self or others (e.g., Ylvisaker & Feeney, 2002), and have therefore been previously implicated in both substance abuse and antisocial behaviour (Ogilvie et al., 2011). Prisoners or people with history of violent or aggressive behaviour tend to report or score lower on neuropsychological measures of executive functioning (Chitsabesan et al., 2015; Morgan & Lilienfed, 2000), and often present with ADHD symptoms (Ginsberg et al., 2010; Meijers et al., 2015). Although this evidence hints that executive function problems and/or ADHD symptoms may play an important role in explaining the pathway between paediatric TBI and antisocial behaviour, to our knowledge there is no empirical work as of yet integrating paediatric TBI, executive dysfunction and antisocial behaviour within a unified longitudinal framework that can be empirically tested.

Who is at higher risk?

As well as to executive function problems, both TBI and antisocial behaviour have also been associated with a range of shared child- or environmentally-related biopsychosocial risk factors. Children with pre-existing learning and behavioural difficulties have been found to be at higher risk of experiencing a TBI, possibly due to the associated tendency to engage in riskier behaviours and/or to anticipate the consequences of their actions (Brown et al., 1981; Goldstrohm & Arffa, 2005; Klonoff, 1971); these are also risk factors for later antisocial behaviour (Fergusson et al., 2005; White et al., 1990). Environmental stressors such as lower socio-economic background,

parental psychopathology, criminality and substance use have also all been linked to both TBI and antisocial behaviour (see e.g., Loeber, 1982; Gaik et al., 2010; McKinlay, 2010; McKinlay et al., 2014). Based on this, it is possible that problem behaviours observed following TBI might be better explained in terms of pre-injury factors rather than brain damage (McKinlay et al., 2010).

Some studies have accounted for this possibility by including early behavioural difficulties and/or adverse family stressors as control variables in their designs. Typically it has been found that, although the inclusion of such factors can slightly weaken the strength of the association between TBI and problem behaviours, this remains significant (see e.g., Fazel et al., 2011; Kennedy et al., 2017; McKinlay et al., 2014). One major problem with including such variables as confounders is that doing so still provides with relatively little information with respect to the exact nature of their contribution in intersecting the association between paediatric TBI and antisocial behaviour; for example, it is possible that early behavioural problems or adverse risk factors might interact with paediatric TBI by exacerbating the risk of experiencing long-term adverse outcomes; conversely, it possible that the lack of pre-existing behavioural problems and/or a positive upbringing experience might act as protective factors, thereby minimising the negative consequences of TBI.

It is also worth noting that, although there has been previous interest in accounting for early development variables, limited attention has been dedicated to risk factors relative to the adolescence period. Within the general population, both substance abuse and parental monitoring (conceptualised as parental knowledge of adolescents' whereabouts, companions and activities; Fletcher et al., 1995) during adolescence have been shown to be associated with poor impulse control and to strongly influence psychosocial outcomes (e.g., Darling et al., 2006; Dishion & McMahon, 1998) including delinquent and antisocial behaviour (Patterson et al., 1984, 1992). Although there is some previous evidence that within young children with TBI, more permissive parenting styles predict worse social competence and more functional impairments at follow-ups (Yeates et al. 2010; Wade et al., 2016), it is currently unclear how these factors in adolescence might influence the pathway from paediatric TBI to adult antisocial behaviour.

Aims and hypotheses

In summary, the long-term psychosocial consequences of paediatric TBI, and particularly whether this is likely to lead to higher risk of engaging in antisocial activities in adulthood remain poorly understood, with few large-scale prospective studies having examined this so far. The mechanisms through which paediatric TBI might lead to antisocial behaviour also remain to be fully clarified. It has been suggested that, since TBI often leads to damage to the brain frontal lobes, injury to the complex neural pathways comprising these structures might impact negatively on the development

of pivotal skills implicated in emotional and behavioural regulation that have been associated with executive function, such as attention and impulse control, leading to higher risk of problem behaviours. Overall, although the research and clinical interest in both TBI and antisocial behaviour are remarkably high at present, there is still a lack of a unifying framework providing with a holistic understanding of the trajectory linking these two factors and relative contributions of key biological, cognitive and social variables (Aguiar, 2016).

The aim of the present research is to build upon such gaps in the literature, and develop and test a novel, time-graded model integrating paediatric TBI, difficulties with attention and impulse control, antisocial behaviour, and a range of child- and parent-based variables important for positive development. Using secondary data collected from the ALSPAC project, this exploratory study examined outcomes in adulthood for children who had sustained a TBI before the age of twelve and compared them to those who had suffered from an orthopaedic injury or no injury at all. The age of twelve has been previously noted to be a useful cut-off to delimit the end of childhood and start of puberty (Newman & Newman, 1998). Since adolescence is a developmental phase characterised by several biological and psychosocial transitions, with higher vulnerability to experiencing mental illness (Belfer, 2008), it was deemed that using this age criterion would help to elucidate more neatly the impact of childhood TBI on later outcomes.

Several different predictions were made. Firstly, it was hypothesised that paediatric TBI would be found to be associated with engagement in antisocial behaviour at the age of twenty, and such association would be mediated by the presence of difficulties in adolescence with attention and impulsivity, as reported by parents (***Hypothesis 1***; see Figure 2 for a pictorial representation of this hypothesis). Mediation refers to the transmission of the effect of a predictor variable (in this case, paediatric TBI) to a dependent variable (antisocial behaviour) through an intermediary variable (executive dysfunction symptoms); it therefore helps to explain *why* a relationship exists between antecedents and outcomes (see Figure 1 for a pictorial illustration of the concept of mediation).

Figure 1. Rather than a direct causal relationship between the predictor and the outcome variable (Figure 1a, where the pathway "c" represents the direct effect of the predictor on the outcome), a mediation model (Figure 1b) proposes that the predictor variable influences the mediator variable (pathway a), which in turn influences the dependent variable (pathway b). The indirect, mediation effect (c') is the by-product of paths a and b.

Figure 1a. Unmediated model.

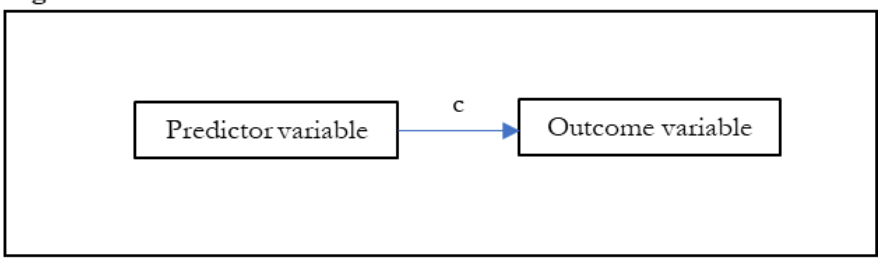


Figure 1b. Basic mediation model.

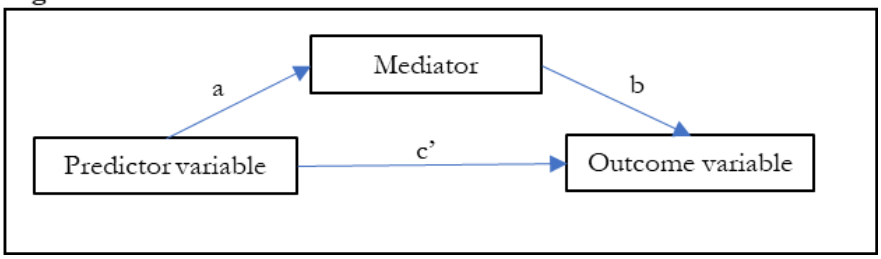
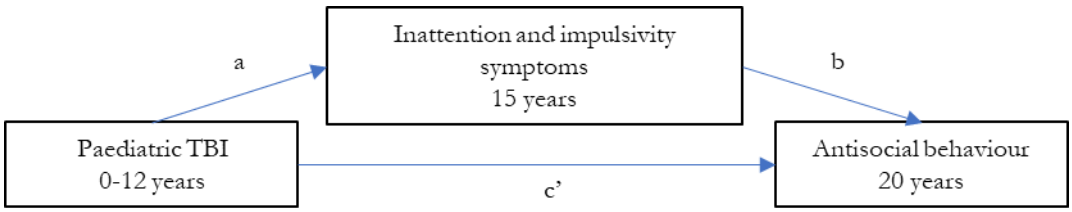


Figure 2: Basic mediation model as predicted by Hypothesis 1, whereby the presence of inattention and impulsivity symptoms would mediate the association between paediatric TBI and antisocial behaviour in adulthood, and therefore explain their relationship.



Secondly, it was hypothesised that the strength of the hypothesized indirect (mediation) effect of paediatric TBI on antisocial behaviour through inattention and impulsivity symptoms would be conditional upon different child- and parent-related moderators. Moderators are variables affecting the direction or strength of a relation between an independent and outcome variable, and thus help to specify *how* or *for who* effects occur (Hayes, 2013). In moderated mediation models, mediation and moderation effects are combined, whereby the indirect effect of the predictor (TBI) on the outcome (antisocial behaviour) via a mediator (executive dysfunction symptoms) differs depending on levels of a moderator variable. With respect to the current study, it was hypothesised that different child- and parent-based variables relative to both the childhood and adolescence period would act as moderators in influencing the indirect association between paediatric TBI and antisocial behaviour. In particular, it was predicted that early emotional and behavioural difficulties (***Hypothesis 2***) and family adversity (***Hypothesis 3***) would moderate the relationship between

paediatric TBI and inattention and impulsivity symptoms, whereby the indirect association between TBI and antisocial behaviour would only be significant for participants who presented with high levels of these compared to those presenting with low levels of them (Figure 3 and 4 below).

Figure 3: Moderated mediation model as predicted by Hypothesis 2, whereby the presence of early emotional and behavioural problems would moderate the indirect association between paediatric TBI and antisocial behaviour in adulthood.

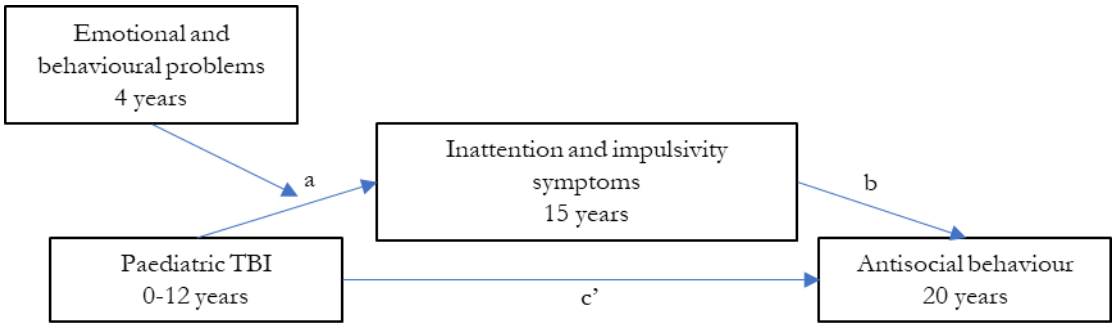
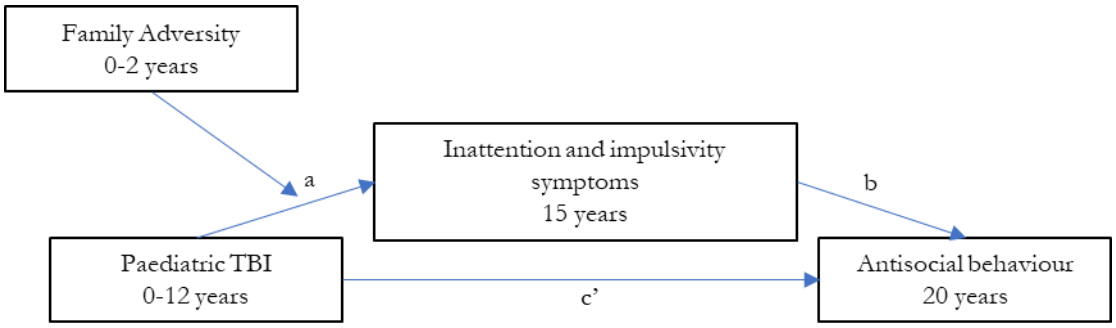


Figure 4: Moderated mediation model as predicted by Hypothesis 3, whereby the presence of family adversity in early childhood would moderate the indirect association between paediatric TBI and antisocial behaviour in adulthood.



With respect to the adolescence period, it was predicted that substance use *(**Hypothesis 4**)* and parental monitoring *(**Hypothesis 5**)* would moderate the relationship between inattention and impulsivity symptoms and antisocial behaviour, whereby the indirect association between paediatric TBI and antisocial behaviour would only be significant for participants with high and low levels of these respectively (Figure 5 and 6 below).

Figure 5: Moderated mediation model as predicted by Hypothesis 4, whereby substance use in adolescence would moderate the indirect association between paediatric TBI and antisocial behaviour.

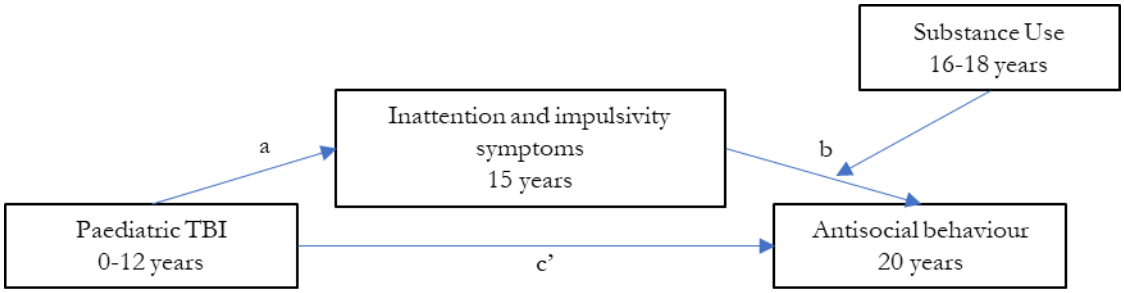
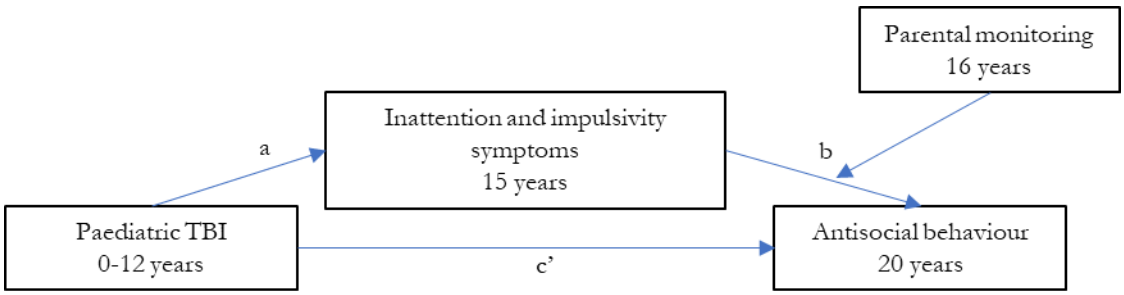


Figure 6: Moderated mediation model as predicted by Hypothesis 5, whereby parental monitoring in adolescence would moderate the indirect association between paediatric TBI and antisocial behaviour in adulthood.



3. Methods

3.1 Participants

Participants were recruited from the Avon Longitudinal Study of Parents and Children (ALSPAC). ALSPAC is an ongoing population-based study investigating the effects of a wide range of genetic and environmental factors on children's development and health. It originally recruited 14,541 pregnant women living in in the former Avon Health Authority in south-west England, all with an expected delivery date between 1991 and 1992, resulting in 13,988 singletons or twins who were still alive at one year of age (Boyd et al., 2012; Fraser et al., 2012). Demographic information regarding the ALSPAC participants was compared with the 1991 national census data, showing that the ALSPAC sample is broadly representative of the UK population (Boyd et al., 2013). It is worth noting that the ethnic composition of the sample is predominantly White (96.06%), which is consistent with the Avon area at the time of recruitment. The ALSPAC official website contains more detailed information regarding the project, including a fully searchable data dictionary with details of all available data collected: www.bris.ac.uk/alspac. Ethical approval for the present study was granted both by the ALSPAC Law and Ethics Committee as well as the local research ethics committees.

3.2 Variables

3.2.1 Prenatal and childhood variables

3.2.1.1 Family Adversity Index (FAI)

Family-based risk factors were evaluated and collated using the Family Adversity Index (FAI; Bowen et al., 2005), assessed both during pregnancy (18–32 weeks gestation) and post-natally between 0 and 2 years. The pre- and post-natal FAI indices were developed based on Rutter's original indicators of adversity (1978, 1979) and have been adopted by several studies using the ALSPAC data to measure environmental risk factors (e.g. Barker et al., 2011). Both FAI indices were based on seventeen family-based risk factors across eight domains (age of mother, housing adequacy, no educational qualifications, financial difficulties, poor partner relationships, maternal substance abuse; maternal criminal behaviour). Each risk factor was rated 1 if adversity was present, with scores summed to create a scale.

3.2.1.2 Early emotional and behavioural difficulties

Emotional and behavioural difficulties were assessed at the age four by administering the parent-based version of the Strengths and Difficulties Questionnaire (SDQ; Goodman, 2001), completed

by participants' mothers or main carers. The SDQ has been previously shown to have good validity and reliability (Goodman, 2001), and is widely used in both research and clinical settings. It comprises a total of 25 items, scored on a scale from 0 to 2 (where *0=not true, 1=somewhat true,* and *2=certainly true*). 20 out of the 25 items can be added together to generate a "total difficulties" score. Total difficulties scores were computed for all participants and then converted into binary indicators (where *0=not high risk* and *1=high risk*), based on previously established cut-off norms developed for 5-to-10 years-old children from England and Wales (Meltzer et al., 2000).

3.2.1.3 TBI and orthopaedic injury

TBI was examined via parental report. When the study children were 4.5, 5.4, 6.4, 8.5, and 11.7 years of age, their mothers or main carers completed questionnaires examining whether they had sustained different types of injuries since birth until time of testing. Participants were assigned to the "TBI group" if a positive response was given to questions asking whether participants had ever sustained a head injury leading to "loss of consciousness", or if they had ever broken or cracked their skull. They were assigned to the "orthopaedic injury" group if they did not report a TBI but provided a positive response to questions asking whether they ever had broken an "arm/hand", "leg/foot", or "any other bone". Participants who suffered from both a TBI and an orthopaedic injury were assigned to the TBI group only. Individuals with no history of TBI or orthopaedic injury were assigned to the "no injury" group.

3.2.2 Adolescence variables

3.2.2.1 Inattention and impulsivity symptoms

Difficulties with attention and impulsivity were assessed using the computerised version of the Development and Well Being Assessment (DAWBA), completed by participants' mothers or main carers at the age of 15.5 years old. The DAWBA is a well-validated semi-structured interview originally developed for the British Child Mental Health surveys (Meltzer et al., 2000). It assesses symptoms and difficulties relating to common emotional and behavioural disorders, with questions relating closely to established diagnostic criteria. The DAWBA also enquires about deficits in attention, impulse control and hyperactivity, which underpin attention-deficit disorders such as ADHD. Since the present research was predominantly interested in elucidating the role of difficulties that are likely to relate to executive function deficits, only total scores for symptoms relative to attention and impulsivity were used. These were added up to create a total score, with higher scores reflecting more significant symptoms. It is worth noting that difficulties around impulsivity were also measured by ALSPAC by administering a neuropsychological measure of

this (the Stop Signal task) at the age of fifteen; however, this was associated with a ceiling effect in performance. Subsequent different manipulations were made to the task during the testing process, making the data difficult to interpret and use (for more information please see the ALSPAC data dictionary, www.bris.ac.uk/alspac).

3.2.2.2 Substance use

Tobacco, cannabis, and alcohol use was assessed at the ages of 16 and 18 years. At each timepoint, participants completed written questionnaires enquiring about the frequency of use of tobacco and cannabis (from "never" to "daily"). Alcohol use was examined using the Alcohol Use Disorders Identification Test (AUDIT; Babor et al., 2001). Participants' responses to individual items were summed up to create total scores for tobacco, cannabis and alcohol use at each time point (resulting in six total scores, with higher scores reflecting more frequent use). Principal component analysis (PCA) with varimax rotation was then conducted with the obtained scores as a dimension reduction technique to compute composite scores reflecting adolescent substance use, using SPSS Statistics version 24. The Kaiser-Meyer-Olkin measure of sampling adequacy was .739, above the recommended value of .6, and Bartlett's test of sphericity was significant ($\chi 2$ (15) = 6571.14, p<.0001). The diagonals of the anti-image correlation matrix were all over .5, supporting the inclusion of each score in the factor analysis, and communalities were all above .3, confirming that each item shared common variance with the others. The PCA yielded only one component with an eigenvalue >1; a single-factor solution was thus retained. This had an eigenvalue of 3.52, explaining 59% of the variance, with factor loadings ranging from .560 to .838.

3.2.2.3 Parental monitoring

Parental monitoring in adolescence was assessed by Likert-type items included in questionnaires completed by the participants' mothers or main carers at the age of 16.5 years. These enquired about the frequency the respondent knew the following (from "never" to "always"): *what their child was doing in their spare time; where they were going when they went out; who they were going out with; what time they would be home.* Responses to these questions were summed up to create a total score, with higher values reflecting lower levels of monitoring.

3.2.3 Adulthood variables

3.2.3.1 Antisocial behaviour

Antisocial behaviour was evaluated via a self-report questionnaire completed by participants at the age of twenty years. This included a total of twelve questions, enquiring about the number of times

in the previous year that respondents had engaged in behaviours such as vandalism, theft, and violence (e.g., "*How often in the last year have you: hit, kicked or punched someone else with the intention of really hurting them?… stolen something from a shop or store?").* Participants were provided with four response options: "not at all", "once", "two-five times", and "six or more times". These were assigned a score of one, two, three and four respectively; scores were summed up across items to create a total score, with higher values reflecting higher levels of antisocial behaviour.

3.2.3 Statistical Analyses

Analyses were performed using Mplus version 8. Missing data were accounted for by full information maximum likelihood estimation. In our main analyses, participants with TBI were compared to all those with no history of TBI; the independent variable (history of TBI) was dummy-coded, with individuals reporting no history of TBI as the reference group. Subsequently, sensitivity analyses were conducted comparing participants with history of TBI versus those with orthopaedic injuries, as well as participants with history of orthopaedic injuries versus those with no history of any injury. To assess model fit, the following indices were adopted: Comparative Fit Indices (CFI), the Tucker-Lewis Index (TLI), and the Root Mean Square Errors of Approximation (RMSEA), recommended by previous research, and all generated automatically by Mplus ouputs. CFI and TLI values higher than .95 and RMSEA values lower than .05 indicate adequate fit (Hu & Bentler, 1999).

Different approaches have been used in the literature to test mediation and moderated mediation effects. The use of bootstrapping procedures with confidence intervals has been strongly advocated (Hayes, 2013; MacKinnon et al., 2004). One main strength of bootstrapping procedures compared to other approaches is that they do not assume indirect effects to be normally distributed; this is particularly important since standard errors underlying indirect effects are typically skewed (Edwards & Lambert, 2007). Thus the use of bootstrapping in the present study all indirect effects were estimated using bootstrapping (samples=10,000). The 2.5 and 97.5 percentiles of the empirical sampling distribution were used to form a 95% confidence interval (CI). If the 95% CI does not contain zero at the selected level of confidence ($p<.05$), the Null Hypothesis can be rejected and the result is considered statistically significant (Preacher & Hayes, 2008).

The analyses proceeded in three main steps. In step 1, the relationship between paediatric TBI and antisocial behaviour in adulthood and mediating role of executive dysfunction symptoms in adolescence was examined (Model 1). Then, proposed childhood moderators (i.e., SDQ and FAI)

of the pathway linking paediatric TBI and executive dysfunction symptoms in adolescence were added to the original model to test for moderated mediation (Model 2 and 3 respectively). In step 2, nonsignificant paths were removed, and substance use and parental monitoring in adolescence were added as moderators of the pathway linking executive dysfunction symptoms and antisocial behaviour (Model 4 and 5 respectively). In step 3, a new revised model integrating the findings from Step 1 and 2 was developed and tested (Model 6).

4. Results

4.1 Descriptive Statistics

Tables 1 and 2 show the descriptive statistics and correlations among key study variables obtained prior to testing for any model.

4.2 Main analyses

4.2.1 Step 1 analyses

4.2.1.1 Model 1: Basic mediation

Model 1 hypothesised that the association between paediatric TBI and antisocial behaviour in adulthood would be mediated by the presence of parent-reported inattention and impulsivity symptoms in adolescence (Figure 2 above). Gender, pre-natal and post-natal FAI were controlled for. Since this model was saturated (i.e. had 0 degrees of freedom), it displayed optimal fit, *CFI* = 1, *TLI* =1, *RMSEA* = 0. As noted in Table 3, the 95% CI for indirect effect of TBI on antisocial behaviour through inattention and impulsivity symptoms did not include 0, supporting the hypothesis. It is worth noting that TBI was found to be a significant predictor of inattention and impulsivity symptoms in adolescence, and these were a significant predictor of antisocial behaviour in adulthood; however, the direct, unmediated effect of TBI on antisocial behaviour was not significant.

In the sensitivity analyses comparing the TBI group with those with orthopaedic injury, TBI was still found to be more strongly associated with inattention and impulsivity symptoms than orthopaedic injury; however, the mediation effect was weakened and no longer significant (see supplementary tables in Appendix B). In the sensitivity analyses comparing the orthopaedic injury group with those with no injury, there were no significant direct effects of orthopaedic injury or

mediation effect, suggesting that the orthopaedic and no injury groups did not differ significantly in terms of outcomes (see supplementary tables in Appendix C).

Table 1: *Descriptive statistics for key variables.*

	Variables							
Group	Gender (% male)	FAI pre-natal	FAI post-natal	SDQ	Inattention and impulsivity	Substance use	Parental monitoring	Antisocial behaviour
TBI *(n=403)*	59.06	1.45 (1.68)	2.28 (2.17)	.09 (.28)	3.68 (5.55)	-.10 (.90)	6.95 (2.09)	12.71 (1.41)
Orthopaedic *(n=1382)*	50.72	1.07 (1.42)	1.71 (1.89)	.06 (.25)	2.55 (4.46)	-.16 (.91)	6.56 (2.11)	12.57 (1.31)
No injury *(n=5398)*	49.43	1.03 (1.40)	1.70 (1.85)	.05 (.22)	2.60 (4.38)	-.22 (.90)	6.45 (2.13)	12.48 (1.23)

Note. TBI=Traumatic Brain injury; FAI=Family Adversity Index; SDQ=Strengths and Difficulties Questionnaire

Table 2: *Intercorrelations among key variables.*

Variables	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**	**7.**	**8.**
1. TBI	-	.070**	.074**	.039*	.070**	.044	.055*	.040
2. FAI pre-natal	.070**	-	.607**	.151**	.211**	.160**	.077**	.107**
3. FAI post-natal	.074**	.607**	-	.145**	.183**	.124**	.117**	.089**
4. SDQ	.039*	.151**	.145**	-	.228**	.044	.055*	.100**
5. Inattention and impulsivity	.070**	.211**	.183**	.228**	-	.199**	.166**	.202**
6. Substance use	.044	.160**	.124**	.044	.199**	-	.366**	.369**
7. Parental monitoring	.055*	.077**	.117**	.055*	.166**	.366**	-	.178**
8. Antisocial behaviour	.040	.107**	.089**	.100**	.202**	.369**	.178**	-

Note. TBI=Traumatic Brain injury; FAI=Family Adversity Index; SDQ=Strengths and Difficulties Questionnaire
**p<.001 *p<.01

Table 3: Results of Model 1 (basic mediation model).

	Inattention and impulsivity *(Mediator)*				Antisocial behaviour *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct effects								
TBI *(predictor)*	.040	.869	.424	[.082, 1.746]	.020	.123	.127	[-.107, .397]
Inattention and impulsivity *(mediator)*					.163	.046	3.661	[.024, .074]
Indirect effects								
TBI on antisocial behaviour through inattention and impulsivity					.006	.040	.023	[.007, .101]
Total effects								
TBI on antisocial behaviour					.026	.163	.127	[-.063, .438]

Note. TBI=Traumatic Brain injury; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap samples.

4.2.1.2 Model 2: Moderated mediation with the SDQ

Model 2 hypothesised that the indirect association between paediatric TBI and antisocial behaviour through inattention and impulsivity symptoms would only be significant for those scoring highly on the SDQ (Figure 3). Gender, pre-natal and post-natal FAI were controlled for. The model had adequate fit, *CFI* =.990, *TLI* =.934, *RMSEA*= .022, 90% CI =.004, .042. As noted in Table 4, the 95% CI for the interaction effect between TBI and SDQ did not include 0, indicating significant moderated mediation by the SDQ. Table 4 also displays the conditional indirect effect of TBI on antisocial behaviour at the two values of the SDQ (0 and 1). The 95% CI for the conditional indirect effect contained 0 at the low value of the SDQ, but did not contain 0 at its high value. This result indicates a significant conditional indirect effect of TBI on antisocial behaviour when participants score high (but not low) on the SDQ, supporting the hypothesis.

In the sensitivity analyses comparing the TBI group with those with orthopaedic injury, the moderated mediation effect remained significant; however, the conditional indirect effect of TBI on antisocial behaviour at the high value of the SDQ was no longer significant (Appendix B). In the sensitivity analyses comparing the orthopaedic injury group with those with no injury, the moderated mediation effect was not significant (Appendix C). Taken together, these results suggest that the moderated mediation effect holds when people with TBI are compared to the rest of the population, but is weakened in strength when they are compared with people with orthopaedic injury. Since, however, individuals with orthopaedic injury did not differ from those with no history of any injury, orthopaedic injury alone or in interaction with early behavioural and emotional problems does not appear to be a significant risk factor for the outcomes examined.

Table 4: Results of Model 2 (moderated mediation model).

	Inattention and impulsivity *(Mediator)*				Antisocial behaviour *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct and interaction effects								
TBI *(predictor)*	.016	.360	.371	[-.331, 1.146]	. 017	.105	.132	[-.126, .395]
Inattention and impulsivity *(mediator)*					.163	.046	.013	[.023, .074]
SDQ *(moderator)*	.149	3.090	.500	[2.135, 4.082]				
TBI x SDQ *(interaction effect)*	.086	.390	3.060	[1.432, 13.441]				
Conditional indirect effects at different values of SDQ						**B**	**SE**	**95% CI**
SDQ=0 *(low value)*						.016	.018	[-.012, .063]
SDQ=1*(high value)*						.362	.174	[.093, .786]
Total effects at different values of SDQ								
SDQ=0 *(low value)*						.122	.132	[-.107, .409]
SDQ=1 *(high value)*						.467	.215	[.091, .944]

Note. TBI=Traumatic Brain injury; SDQ=Strengths and Difficulties Questionnaire; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap sample.

4.2.1.3 Model 3: Moderated mediation with the FAI

Model 3 hypothesised that the indirect association between paediatric TBI and antisocial behaviour through inattention and impulsivity symptoms would only be significant for those with high scores on the post-natal FAI (Figure 4). Gender, pre-natal FAI, and SDQ were controlled for. The model had adequate fit, *CFI*=1, *TLI*=1.024, *RMSEA*=.000, 90% CI =.000, .018. As noted in Table 5 (see Appendix A for supplementary tables), the 95% CI for the interaction effect between TBI and FAI included 0, indicating that moderated mediation effect by the FAI was not significant. The hypothesis was thus not supported and FAI was not retained as a moderator in subsequent models.

The sensitivity analyses comparing the TBI group with those with orthopaedic injury, and the orthopaedic injury group with the no injury group yielded similar results, as the moderated mediation effect by the FAI was not significant in either case (Appendices B and C).

4.2.2 Step 2 analyses

4.2.2.1 Model 4: Moderated mediation by substance use

Model 4 hypothesised that the indirect association between paediatric TBI and antisocial behaviour through inattention and impulsivity symptoms would only be significant for those with high levels of substance use (Figure 5). The model had inadequate fit, *CFI* =.899, *TLI* =.573, *RMSEA*= .066, 90% CI =.041, .093. The model was thus not explored further (see Appendix A for supplementary tables). The sensitivity analyses comparing the TBI group with those with orthopaedic injury, and the orthopaedic injury group with the no injury group also yielded a similar result (Appendices B and C).

4.2.2.2 Model 5: Moderated mediation by parental monitoring

Model 5 hypothesised that the indirect association between paediatric TBI and antisocial behaviour through inattention and impulsivity symptoms would be stronger for those with low levels of parental monitoring (Figure 6). The model had a poor fit, *CFI* =.065, *TLI* =-2.974, *RMSEA*= .744. 90% CI =.729, .758. The model was thus not explored further (Appendix A). The sensitivity analyses comparing the TBI group with those with orthopaedic injury, and the orthopaedic injury group with the no injury group also yielded a similar result (Appendices B and C).

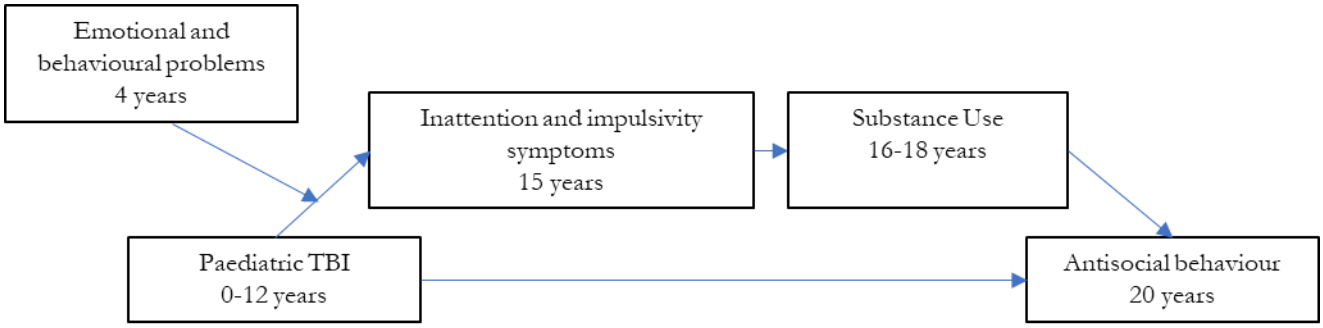
4.2.3 Step 3

4.2.3.1 Exploratory analyses

Following the findings from Models 4 and 5, revealing that inclusion of substance use and parental monitoring in adolescence led to poor-fitting models, previous literature was reviewed and re-examined to build an alternative theoretical framework. The focus was predominantly on trying to elucidate the possible contribution of substance use in adolescence since there is more evidence linking this to both TBI and antisocial behaviour compared to parental monitoring.

An alternative hypothesis regarding the potential contribution of substance use is that this plays a mediating (rather than moderating) role in explaining the indirect relationship between paediatric TBI and antisocial behaviour. There is evidence indicating that young individuals with executive function difficulties and/or ADHD diagnoses are more likely to engage in risk-taking behaviours such as substance use, possibly due to their low ability to regulate behaviour and consider the consequences of their actions (Dolan et al., 2008; Verdejo-Garcia et al., 2006). Substance use, in turn, is known to also lower people's ability to plan, control and inhibit their own thoughts and behaviours, consequentially increasing the likelihood that people might engage in antisocial and/or criminal acts (Brady et al., 1998). McKinlay et al. (2014), in their prospective study on a population of inpatients and outpatients in New Zealand, found that when they accounted for history of alcohol and drug dependence during adolescence and early adulthood by adding this as a covariate in the study, there were no longer significant associations between TBI before the age of five and offending; the authors suggested that this may be evidence that substance use mediates the association between the two variables. Similarly, Kennedy et al. (2017) found that adjustment for substance use in their analyses weakened the associations between TBI and offences and trouble with the police at seventeen years of age. Based on this, it was hypothesized that substance use, in addition to inattention and impulsivity symptoms, might also lie on the pathway linking TBI and antisocial behaviour, and contribute to explain their relationship (see Figure 6 below). To test this idea, in model 6 substance use was added as a second, serial mediator.

Figure 7. Pictorial representation of Model 6 (moderated serial mediation).



4.2.3.2 Model 6: moderated serial mediation

In model 6 it was hypothesised that both inattention and impulsivity symptoms and substance use would mediate the indirect association between TBI and antisocial behaviour, and that the indirect effect of TBI on antisocial behaviour through both mediators would be significant only for those scoring highly on the SDQ. The model had adequate fit, *CFI* =.970, *TLI* =.909, *RMSEA*= .023, 90% CI =.013, .033. As noted in Table 5 below, the 95% CI for the interaction effect between TBI and SDQ did not include 0, confirming significant moderated mediation by the SDQ. Table 5 also displays the conditional indirect effect of TBI on antisocial behaviour through both executive dysfunction symptoms and substance use at the two values of the SDQ. The 95% CI for the conditional indirect effect contained 0 at the low value of the SDQ (suggesting no significant conditional indirect effect); however it did not contain 0 at the high value of the SDQ, suggesting a significant conditional indirect effect, and hence supporting the hypothesis.

The sensitivity analyses comparing the TBI group with those with orthopaedic injury yielded similar findings, as the moderated serial mediation effect was significant at the high value of the SDQ (Appendices B). This was not significant in the sensitivity analyses comparing the orthopaedic injury and the no injury groups (Appendix C). These findings suggest that the moderated serial mediation effect identified in Model 6 is specific to those presenting with a TBI.

Table 5: Results of Model 6 (moderated serial mediation model).

	Inattention and impulsivity *(Mediator 1)*				Substance use *(Mediator 2)*				Antisocial behaviour *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct and interaction effects												
TBI *(predictor)*	.016	.356	.369	[-.332, 1.126]	.028	.132	.127	[-.103, .396]	.003	.018	.136	[-.230, .309]
Inattention and impulsivity *(mediator 1)*					.147	.033	.010	[.015, .053]	.108	.029	.012	[.007, .056]
SDQ *(moderator)*	.148	3.080	.500	[2.142, 4.076]								
TBI x SDQ *(interaction effect)*	.085	7.470	3.058	[1.295, 13.436]								
Substance use *(mediator 2)*									.339	.476	.075	[.336, .629]
Conditional indirect effects										**B**	**SE**	**95% CI**
Via inattention and impulsivity												
SDQ=0 *(low value)*										.011	.013	[-.007, .047]
SDQ=1 *(high value)*										.231	.137	[.042, .611]
Via substance use										.063	.062	[-.045, .202]
Via both mediators, given values of SDQ												
SDQ=0 *(low value)*										.006	.006	[-.004, .023]
SDQ=1 *(high value)*										.122	.063	[.031, .292]
Conditional total effects												
SDQ=0 *(low value)*										.097	.130	[-.136, .380]
SDQ=1 *(high value)*										.433	.213	[.082, .929]

Note. TBI=Traumatic Brain injury; SDQ=Strengths and Difficulties Questionnaire; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap samples

5. Discussion

Using an epidemiological birth cohort, the present exploratory study aimed to examine the relationship between paediatric TBI, antisocial behaviour, and a range of key child- and environmentally-related psychosocial variables. Consistent with our first hypothesis (*Hypothesis 1*), parent-reported symptoms relative to attention and impulsivity in adolescence mediated the association between TBI before the age of twelve and antisocial behaviour at the age of twenty. Moreover, as predicted *(Hypothesis 2),* early childhood emotional and behavioural difficulties moderated the pathway from paediatric TBI to inattention and impulsivity symptoms, whereby the indirect association between TBI and antisocial behaviour was significant only for individuals with early emotional and behavioural difficulties. In contrast with our original hypotheses, however, early family adversity was not a significant moderator *(Hypothesis 3).* Our models predicting that substance use and poor parental monitoring in adolescence would moderate the association between inattention and impulsivity symptoms and antisocial behaviour (*Hypothesis 4 and 5 respectively*) were found to be a poor fit to the data. A revised theoretical framework was then developed based on these findings and revision of previous literature (*Model 6, Figure 7 above).* It was found that both inattention and impulsivity symptoms and substance use serially mediated the association between TBI and antisocial behaviour, which continued to be moderated by the presence of early emotional and behavioural problems.

The present study helps to enhance our understanding of the long-term consequences of TBI, past the puberty period. By including a number of key psychosocial factors previously associated with increased risk for both TBI and antisocial behaviour within a single framework, the present research has the potential to better elucidate the nature of the pathways leading from TBI to antisocial behaviour, and gain more insight into "how", "why", and "for who" this might be a risk factor for adverse outcomes later in life. Taken together, the current findings suggest that children with history of paediatric TBI before the age of twelve are at significant risk of presenting with parent-reported executive function symptoms in adolescence around attention and impulsivity. Such executive function problems, in turn, appear to predict an increased risk for substance use, leading to increased chance of engaging in antisocial behaviour in adulthood. Both executive dysfunction symptoms and substance use thus form a developmental chain linking TBI with antisocial behaviour (serial mediation, Hayes, 2013). Notably, this pattern appeared to be true only for children with early emotional and behavioural difficulties; those who suffered from a TBI but did not have such issues were not found to be at the same risk of adverse outcomes in adolescence or adulthood.

The mediating role of executive function difficulties and substance use

A number of key considerations can be made from the present findings. The finding that TBI predicted more executive dysfunction symptoms, leading to higher likelihood of using substances in adolescence and eventually engaging in antisocial acts, helps to shed some light on why TBI might lead to criminal behaviour. TBI has been previously found to be associated with impaired neuropsychological functioning; difficulties around key executive function skills such as attention and impulse control are common in children following a TBI (Sinopoli et al., 2011). In the uninjured population, executive function problems have been implicated in poor decision-making, sensation-seeking and engagement in reckless acts causing risk to self or others, including both substance use and aggression (Krämer et al., 2011). There is previous evidence that, when accounting for substance use, the association between TBI and offending either weakens or is no longer significant (Kennedy et al., 2017; McKinlay et al., 2014). The present study expands on previous literature by indicating that both executive function problems and substance use indeed mediate the association between paediatric TBI and antisocial behaviour thereby explaining why these two might be connected. This is the first study to our knowledge providing direct evidence for a developmental pathway among these variables within a single, time-graded framework.

Family- and child-based biopsychosocial moderators

Interestingly, whereas early emotional and behavioural difficulties moderated the indirect association between TBI and antisocial behaviour (by influencing the likelihood that individuals would develop executive dysfunction symptoms), family adversity was not found to have such role. Moreover, although TBI correlated with lower levels of parental monitoring in adolescence, inclusion of the latter as a moderator led to the model being a poor fit to the data. Taken together, these findings suggest that child-related variables such as difficult early emotional and behavioural problems might play a more pivotal role in influencing the pathway from paediatric TBI to antisocial behaviour than environmental influences. What could be possible reasons for this? A potential explanation is that environmentally-related, psychosocial factors such as family adversity might have a less determinant impact than individual characteristics on influencing the likelihood of developing ADHD symptoms related to attention and impulsivity. Conversely, family environment might play a more important role in influencing psychosocial outcomes that are less reliant on central nervous system integrity. Indeed, evidence can be found in the literature on TBI hinting towards such possible dissociation regarding the role of individual versus environmental variables (Taylor et al., 2002; Yeates et al., 1997, 2010). For example, Yeates et al. (2002) found no moderation effect of family environment in influencing neuropsychological outcomes in children

at 6-, 12-month and 4-year post-injury follow-ups, whereas other studies have shown strong association between quality of family interactions and internalizing difficulties, social competence and behavioural adjustment following a TBI (Lezak, 1978; Worthington, 1989). It is thus possible that the relative influence of different biopsychosocial factors depends on the type of outcome examined, a suggestion that could be explored in more detail by future research. Another possibility is that the present measure of environmental adversity was not sufficiently sensitive, and/or did not include family factors that have a more important role in affecting the link between TBI and problems with attention and impulsivity. For instance, there is previous evidence that child maltreatment and abuse are strong predictors of altered cognitive development (see e.g., De Prince et al., 2009; Mezzacappa et al., 2001). Brewer-Smyth et al. (2015) found that, within an inmate population with history of paediatric TBI, TBI became negatively associated with committing a violent crime after accounting for history of stressful childhood events such as sexual and emotional abuse; factors such as history of trauma, neglect and abuse might therefore play a more pivotal influence than those included in the current study in intersecting the pathway between TBI and antisocial behaviour. This potential limitation could be overcome by future studies by measuring and including different types of variables.

TBI vs orthopaedic injury: differences in outcomes?

One additional strength of this study was the inclusion of a negative exposure control group comprising individuals who had suffered from an orthopaedic injury that did not involve the skull. Orthopaedic injury has a similar confounding structure to TBI but no plausible biological connection to antisocial behaviour; comparing outcomes between people with TBI versus those with orthopaedic injury therefore helps to ascertain whether the association is more likely to be explained by potentially unobserved or unaccounted for confounding biases (e.g., the traumatic experience of suffering from any type of injury; see also Gage et al., 2016 for a discussion of the utility of including negative exposure control groups). With respect to the present study, it is interesting to note that, when the group with TBI was compared to those with orthopaedic injury, although the association between TBI and the outcomes of interest was slightly weakened, it remained significantly stronger for those with history of TBI. Moreover, when the orthopaedic injury and the no injury control groups were compared, the moderated serial mediation effect was no longer significant. Taken together, these findings suggest that the effects identified in the study are specific to TBI as opposed to suffering from any type of injury. It is interesting though that these findings do not fully support the previous study by Kennedy et al. (2017) using the same ALSPAC population, who found that people with a TBI and orthopaedic injury were at similar

odds of criminal behaviour at the age of seventeen. Such discrepancy in the findings might be due to different reasons. Firstly, it is worth noting that when TBI was considered in isolation, the association between this and antisocial behaviour was not found to be stronger than for orthopaedic injury; inclusion of early emotional and behavioural problems in the model was crucial for elucidating under which circumstances TBI was a significant risk factor compared to orthopaedic injury; this might explain why in our research we were able to identify specific effects relative to TBI that were not evident in the study by Kennedy et al. Secondly, it is also possible that both people with TBI and orthopaedic injury are both in fact more likely to present with adverse outcomes during adolescence, possibly due to shared characteristics such as sensation-seeking. Since adolescence is a developmental stage characterized by higher risk of externalizing difficulties, this might exacerbate likelihood of showing problem behaviours for more vulnerable populations such as those with history of TBI and orthopaedic injury. However, it might be that whereas such risks become attenuated for people with orthopaedic injury as puberty comes to an end, these might still remain significantly higher for those with TBI compared to the uninjured population. Future research might elucidate trajectories for people with TBI versus orthopaedic injury further by comparing outcomes at different life points and examine the influence of possible shared confounding factors such as risk-taking tendencies.

5.1 Implications

The present findings have the potential to advance current understanding of the link between paediatric TBI and antisocial behaviour. The study presents with a novel theoretical framework that helps to elucidate the nature of the relationship between these two factors over the time, as well as the role of key biopsychosocial factors. Previous authors have recently highlighted the lack of a unifying framework for understanding TBI and need to understand this in a more holistic manner (Aguiar, 2016). The findings highlight that inclusion of key individual biopsychosocial factors is pivotal for gaining a better understanding of what type of individuals are at higher risk of presenting with adverse outcomes following a paediatric TBI, and through which causal mechanisms. They show that TBI in isolation appears unlikely to be captured as a risk factor for adverse outcomes, thereby highlighting the importance for future research to include early emotional and behavioural problems to ensure to identify the potential long-term risks of TBI.

The findings of the present study also have implications for informing prevention and rehabilitation measures. They indicate that children with paediatric TBI with early conduct and emotional issues comprise a more vulnerable set of the population who is at higher risk of showing

patterns of difficulties with attention and impulse control typically associated with ADHD diagnoses in adolescence, and increased likelihood of engaging in harmful behaviours to the self and others such as substance use and antisocial acts. They indicate that screening for early emotional and behavioural difficulties when individuals present to health services following a TBI might play a fundamental role in helping clinicians to identify which children might be at higher risk of developing future problems. Such children might in turn benefit from more frequent follow-up assessments and reviews over time to monitor their functioning, prevent adverse outcomes and provide additional support where needed. Families and carers might also benefit from more information regarding the possible risks associated with paediatric TBI and means of supporting their offspring in the future. Similarly, the findings also indicate that within adults showing elevated levels of antisocial or criminal behaviours such as for example offenders, screening for history of TBI might help to identify whether such individuals might be presenting with unmet needs, and understand the possible mechanisms leading to their problem behaviours; rehabilitation goals and interventions could then be tailored accordingly.

5.2 Limitations and future directions

Although the present study presents with a number of methodological strengths, it is not free of limitations. Firstly, all variables of interest were measured via self-report. There is previous evidence that people can often fail to remember previous history of TBI, especially when this occurred at an early age, leading to inaccuracies in reports (McKinlay et al., 2016). History of antisocial behaviour may also tend to be under-reported due to social desirability and impression management biases (Edens, 2004). Such limitations are intrinsic to the way the data were originally collected and thus could not be avoided; large-scale longitudinal studies are time- and cost-consuming, meaning that that self-report can often represent an efficient way to maximize the amount of information collected. Future studies could overcome such limitations where the resources allow for this, by for example examining clinical and criminal records, and/or collecting information from different informants. The present study also does not provide with information regarding the nature, location, severity and number of TBI(s) that participants suffered from; more details regarding this would be beneficial for drawing more precise conclusion regarding the link between different types of TBI (e.g., mild versus severe) and adverse outcomes.

Another issue of the present research relates to the assessment of attention and impulsivity difficulties in the participants, obtained via parental report. Although it is likely that difficulties with executive function skills are likely to underpin high scores for questions related to inattention

and impulsivity symptoms, based on the present measure it is not possible to establish with certainty whether participants did in fact present with executive function problems Moreover, although the measure used to evaluate the presence of these difficulties, the DAWBA, is a questionnaire of established validity and reliability (Meltzer et al., 2000), it cannot be excluded that parents' reports on this might have been influenced by a variety of factors; for example, parents of children who already had an ADHD diagnosis might have been biased towards reporting more difficulties in these domains. The presence of neuropsychological impairments in relation to attention and impulsivity should be ascertained more rigorously in the future through the use of standardised neuropsychological measures and more thorough clinical assessments of participants' presentations. It is worth noting that the present study also included assessments of only some aspects of executive function (low attention and impulse regulation); executive function is an umbrella term that includes several different abilities, such as for instance problem-solving, planning, self-monitoring (Anderson, 2002). It is important that future research is conducted to clarify the influence of impairments in different executive function skills, particularly those contributing to the "social brain network" (Williams et al., 2015).

The measure used to examine early emotional and behavioural difficulties at the age of four, the SDQ, also contained two questions addressing impulsivity and inattention. Although only two questions out of twenty targeted such domains, it cannot be excluded that presence of early difficulties in these might have led to an overlap between the SDQ and DAWBA measures, collected at the ages of four and fifteen respectively. In particular, since it was not possible to establish participants' exact age at TBI, it is unclear to what extent early difficulties with attention and inhibition might have been the cause or the consequence of TBI, and of problems in these same areas in adolescence as measured by the DAWBA. Future research might be able to overcome such limitations to an extent by for example recording exact age at TBI more rigorously, adopting measures of emotional, behavioural, and functioning with a lesser degree of overlap with one another to reduce confounding effects, and comparing emotional, behavioural and cognitive functioning at more frequent stages in development. It is worth noting that, although the present findings might not allow to draw definite conclusions regarding the exact aetiology of the relationships among the variables included, these are in fact likely to be very complex in real life; the findings still have important implications with respect to helping to illuminate which individuals are at higher risk of adverse outcomes following a TBI and what factors within them and their environment are likely to deserve particular attention within clinical settings.

Interestingly, the present findings hinted that child-related variables might play a more influential role in intersecting the link between paediatric TBI and antisocial behaviour than parent-based factors. Disentangling the relative contribution of individual versus environmental influences in development is challenging, as parents are likely to provide their children with family environments that strongly correlate with their genotypes ("passive gene-environment correlation"; Plomin et al., 1977); the resulting association between the children biological predisposition and home environment then becomes "spurious", meaning that it is difficult to disentangle the exact role of individual versus parental variables on adverse outcomes. In the future twin studies might play a fundamental role in capturing more precisely the extent to which TBI predicts antisocial behaviour independently of latent familial risks, by for example comparing monozygotic and dizygotic twins who grew up in similar versus different family environments.

5.3 Conclusions

In summary, the present study aimed to investigate the link between paediatric TBI and antisocial behaviour in adulthood using the data collected by ALSPAC. The role of key child- and environmentally-related biopsychosocial factors in intersecting this association was also examined. After testing several consecutive theoretical models, it was found that executive dysfunction symptoms relative to low attention and impulse control, and substance use in adolescence mediated the relationship between TBI and antisocial behaviour in a serial fashion. The presence of behavioural and emotional difficulties in early childhood moderated the pathway between TBI and executive dysfunction symptoms, whereby only children with early emotional and behavioural problems were at risk of adverse outcomes later on in life. The present research offers with a novel, time-graded framework that can hopefully represent a step forwards towards gaining a more holistic understanding of the association between paediatric TBI, antisocial behaviour and other key biopsychosocial factors important for positive development. The findings have implications for informing preventative and rehabilitation measures both within the healthcare and criminal justice systems.

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Appendix A: Non-significant or poor fit models

Table 6: Results of Model 3 (moderated mediation model).

	Inattention and impulsivity *(Mediator)*				Antisocial behaviour *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct and interaction effects								
TBI *(predictor)*	.012	.256	.574	[-.801, 1.446]	.019	.118	.132	[-.114, .406]
Inattention and impulsivity *(mediator)*					.150	.042	.012	[.021, .069]
FAI *(moderator)*	.061	.155	.060	[.040, .274]				
TBI x FAI *(interaction effect)*	.034	.269	.249	[-.228, .740]				
Conditional indirect effects at different values of FAI						**B**	**SE**	**95% CI**
FAI=0 *(low value)*						.011	.026	[-.032, .073]
FAI=1 *(medium value)*						.022	.020	[-.007, .075]
FAI=3 *(high value)*						.045	.026	[.006, .116]
Total effects at different values of FAI								
FAI=0 *(low value)*						.129	.133	[-.103, .417]
FAI=1 *(medium value)*						.140	.132	[-.090, .425]
FAI=3 *(high value)*						.162	.133	[-.070, .453]

Note. TBI = Traumatic Brain Injury; FAI = Family Adversity Index; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap sample.

Table 7: Results of Model 4 (moderated mediation model).

	Inattention and impulsivity *(Mediator)*				Antisocial behaviour *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct and interaction effects								
TBI *(predictor)*	.024	.434	.612	[-.632, 1.785]	-.036	-.157	.113	[-.632, 1.785]
Inattention and impulsivity *(mediator)*					.162	.040	.017	[.009, .075]
SDQ *(moderator on pathway a)*	.142	2.586	.795	[1.169, 4.299]				
TBI x SDQ *(interaction effect)*	.044	3.579	5.730	[-5.674, 12.983]				
Substance use *(moderator on pathway b)*					.138	.138	.063	[.029, .278]
Inattention and impulsivity x Substance use *(interaction effect)*					.223	.044	.026	[-.006, .097]
Conditional indirect effects at different values of moderators						**B**	**SE**	**95% CI**
SDQ=0								
Substance use=-.844 *(low value)*						.001	.012	[-.014, .039]
Substance use=-.538 *(medium value)*						.007	.013	[-.007, .055]
Substance use=.205 *(high value)*						.021	.033	[-.023, .124]
SDQ=1								
Substance use=-.844 *(low value)*						.013	.108	[-.154, .339]
Substance use=-.538 *(medium value)*						.067	.127	[-.106, .460]
Substance use=.205 *(high value)*						.198	.315	[-.284, .998]
Conditional total effects at different values of moderators								

SDQ=0			
Substance use=-.844 *(low value)*	-.156	.113	[-.386, .062]
Substance use=-.538 *(medium value)*	-.150	.111	[-.368, .069]
Substance use=.205 *(high value)*	-.136	.109	[-.341, .088]
SDQ=1			
Substance use=-.844 *(low value)*	-.144	.160	[-.447, .193]
Substance use=-.538 *(medium value)*	-.090	.172	[-.379, .316]
Substance use=.205 *(high value)*	.041	.334	[-.505, .826]

Note. TBI = Traumatic Brain injury; SDQ = Strengths and Difficulties Questionnaire; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap sample.

Table 8: Results of Model 5 (moderated mediation model).

	Inattention and impulsivity *(Mediator)*				Antisocial behaviour *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct and interaction effects								
TBI *(predictor)*	.036	.799	.440	[-.018, 1.699]	.005	.037	.174	[-.256, .435]
Inattention and impulsivity *(mediator)*					-.332	-.105	.035	[-.180, -.043]
SDQ *(moderator on pathway a)*	.161	3.406	.566	[2.362, 4.586]				
TBI x SDQ *(interaction effect)*	.080	6.933	3.493	[-.300, 13.424]				
Parental monitoring *(moderator on pathway b)*					.020	.014	.016	[-.018, .045]
Inattention and impulsivity x Parental monitoring *(interaction effect)*					.517	.021	.006	[.011, .034]
Conditional indirect effects at different values of moderators						**B**	**SE**	**95% CI**
SDQ=0								
Parental monitoring=4 *(low value)*						-.016	.014	[-.063, .001]
Parental monitoring=6 *(medium value)*						.018	.012	[.001, .052]
Parental monitoring=8 *(high value)*						.052	.032	[.003, .132]
SDQ=1								
Parental monitoring=4 *(low value)*						-.156	.127	[-.517, .008]
Parental monitoring=6 *(medium value)*						.172	.100	[.024, .434]
Parental monitoring=8 *(high value)*						.500	.255	[.086, 1.105]

Conditional total effects at different values of moderators			
SDQ=0			
Parental monitoring=4 *(low value)*	.021	.176	[-.278, .424]
Parental monitoring=6 *(medium value)*	.055	.175	[-.238, .461]
Parental monitoring=8 *(high value)*	.089	.177	[-.206, .502]
SDQ=1			
Parental monitoring=4 *(low value)*	-.119	.224	[-.551, .324]
Parental monitoring=6 *(medium value)*	.209	.209	[-.138, .694]
Parental monitoring=8 *(high value)*	.537	.313	[.004, 1.246]

Note. TBI = Traumatic Brain injury; SDQ = Strengths and Difficulties Questionnaire; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap sample.

Appendix B: Sensitivity analyses comparing the group with TBI versus those with orthopaedic injury

Table 9: Results of Model 1 (basic mediation model).

Model fit: *CFI* =1.000, *TLI* =1.000, *RMSEA*= .000, 90% CI =.000, .000.

	Inattention and impulsivity *(Mediator)*				**Antisocial behaviour** *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct effects								
TBI *(predictor)*	.071	.863	.436	[.045, 1.770]	.019	.065	.134	[-.179, .350]
Inattention and impulsivity *(mediator)*					.074	.021	.017	[-.010, .058]
Indirect effects								
TBI on antisocial behaviour through inattention and impulsivity					.005	.018	.019	[-.004, .080]
Total effects								
TBI on antisocial behaviour					.024	.083	.136	[-.165, .371]

Note. TBI = Traumatic Brain Injury; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap sample.

Table 10: Results of Model 2 (moderated mediation model).

Model fit*: CFI* =1.000, *TLI* =1.062, *RMSEA*= .000, 90% CI =.000,.048

	Inattention and impulsivity *(Mediator)*				**Antisocial behaviour** *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct and interaction effects								
TBI *(predictor)*	.022	.266	.400	[-.485, 1.079]	.007	.026	.140	[-.231, .325]
Inattention and impulsivity *(mediator)*					.078	.022	.018	[-.010, .058]
SDQ *(moderator)*	.070	1.581	1.200	[-4.449, 4.269]				
TBI x SDQ *(interaction effect)*	.199	8.865	3.530	[2.273, 15.027]				
Conditional indirect effects at different values of SDQ						**B**	**SE**	**95% CI**
SDQ=0 *(low value)*						.006	.012	[-.007, .049]
SDQ=1 *(high value)*						.201	.189	[-.053, .723]
Total effects at different values of SDQ								
SDQ=0						.032	.140	[-.222, .332]
SDQ=1						.226	.247	[-.180, .792]

Note. TBI = Traumatic Brain injury; SDQ = Strengths and Difficulties Questionnaire; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap sample.

Table 11: Results of Model 3 (moderated mediation model).

Model fit: *CFI*=.987, *TLI*=.912, *RMSEA*=.025, 90% CI =.000, .071.

	Inattention and impulsivity *(Mediator)*				**Antisocial behaviour** *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct and interaction effects								
TBI *(predictor)*	.020	.242	.590	[-.869, 1.448]	.017	.059	.141	[-.195, .365]
Inattention and impulsivity *(mediator)*								
FAI *(moderator)*	.024	.066	.119	[-.166, .300]				
TBI x FAI *(interaction effect)*	.064	.267	.262	[-.234, .793]				
Conditional indirect effects at different values of FAI						**B**	**SE**	**95% CI**
FAI=0 *(low value)*						.005	.016	[-.014, .061]
FAI=1 *(medium value)*						.010	.015	[-.005, .065]
FAI=3 *(high value)*						.021	.024	[-.006, .100]
Total effects at different values of FAI								
FAI=0 *(low value)*						.063	.142	[-.188, .372]
FAI=1 *(medium value)*						.069	.142	[-.185, .377]
FAI=3 *(high value)*						.079	.144	[-.178, .396]

Note. TBI = Traumatic Brain injury; FAI = Family Adversity Index; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap sample.

Table 12: Results of Model 4 (moderated mediation model).

Model fit: *CFI*=.903, *TLI*=.589, *RMSEA*=.071, 90% CI =.000, .136.

	Inattention and impulsivity *(Mediator)*				**Antisocial behaviour** *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct and interaction effects								
TBI *(predictor)*	.045	.442	.666	[-.767, 1.862]	-.098	-.269	.161	[-.693, -.022]
Inattention and impulsivity *(mediator)*					.087	.024	.041	[-.063, .079]
SDQ *(moderator on pathway a)*	.113	1.967	1.161	[-.503, 4.111]				
TBI x SDQ *(interaction effect)*	.091	3.783	5.524	[-5.151, 13.666]				
Substance use *(moderator on pathway b)*					.133	.168	.223	[-.174, .665]
Inattention and impulsivity x Substance use *(interaction effect)*					.266	.058	.066	[-.088, .132]
Conditional indirect effects at different values of moderators						**B**	**SE**	**95% CI**
SDQ=0								
Substance use=-.844 *(low value)*						-.011	.023	[-.115, .009]
Substance use=-.538 *(medium value)*						-.003	.014	[-.048, .011]
Substance use=.205 *(high value)*						.016	.042	[-.033, .148]
SDQ=1								
Substance use=-.844 *(low value)*						-.106	.188	[-.846, .099]
Substance use=-.538 *(medium value)*						-.031	.110	[-.372, .111]

Substance use=.205 *(high value)*	.151	.381	[-.338, 1.258]
Conditional total effects at different values of moderators			
SDQ=0			
Substance use=-.844 *(low value)*	-.280	.166	[-.716, -.030]
Substance use=-.538 *(medium value)*	-.272	.162	[-.689, -.026]
Substance use=.205 *(high value)*	-.253	.159	[-.656, -.003]
SDQ=1			
Substance use=-.844 *(low value)*	-.375	.260	[-1.166, -.014]
Substance use=-.538 *(medium value)*	-.300	.197	[-.815, .002]
Substance use=.205 *(high value)*	-.118	.398	[-.725, .958]

Note. TBI = Traumatic Brain injury; SDQ = Strengths and Difficulties Questionnaire; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap sample.

Table 13: Results of Model 5 (moderated mediation model).

Model fit: *CFI*=.075, *TLI*=-2.930, *RMSEA*=.731, 90% CI =.700, .762.

	Inattention and impulsivity *(Mediator)*				**Antisocial behaviour** *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct and interaction effects								
TBI *(predictor)*	.046	.587	.474	[-.315, 1.559]	.006	.022	.182	[-.305, .409]
Inattention and impulsivity *(mediator)*					-.334	-.099	.036	[-.176, -.033]
SDQ *(moderator on pathway a)*	.085	2.020	1.392	[-.394, 5.164]				
TBI x SDQ *(interaction effect)*	.175	8.025	3.716	[.085, 14.938]				
Parental monitoring *(moderator on pathway b)*					-.079	-.055	.027	[-.108, -.003]
Inattention and impulsivity x Parental monitoring *(interaction effect)*					.472	.019	.006	[.009, .033]
Conditional indirect effects at different values of moderators						**B**	**SE**	**95% CI**
SDQ=0								
Parental monitoring=4 *(low value)*						-.015	.019	[-.073, .008]
Parental monitoring=6 *(medium value)*						.007	.013	[-.006, .059]
Parental monitoring=8 *(high value)*						.029	.029	[-.009, .116]
SDQ=1								
Parental monitoring=4 *(low value)*						-.216	.193	[-.714, .073]
Parental monitoring=6 *(medium value)*						.104	.158	[-.109, .566]

Parental monitoring=8 *(high value)*	.424	.278	[.066, 1.211]
Conditional total effects at different values of moderators			
SDQ=0			
Parental monitoring=4 *(low value)*	.007	.186	[-.328, .398]
Parental monitoring=6 *(medium value)*	.029	.185	[-.297, .426]
Parental monitoring=8 *(high value)*	.050	.185	[-.276, .451]
SDQ=1			
Parental monitoring=4 *(low value)*	-.195	.289	[-.767, .371]
Parental monitoring=6 *(medium value)*	.125	.260	[-.294, .747]
Parental monitoring=8 *(high value)*	.445	.340	[-.082, 1.286]

Note. TBI = Traumatic Brain injury; SDQ = Strengths and Difficulties Questionnaire; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap sample.

Table 14: Results of Model 6 (moderated serial mediation model)

Model fit: *CFI* =1.000, *TLI* =1.033, *RMSEA*= .000, 90% CI =.000, .031

	Inattention and impulsivity *(Mediator 1)*				**Substance use** *(Mediator 2)*				**Antisocial behaviour** *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct and interaction effects												
TBI *(predictor)*	.021	.260	.401	[-.493, 1.082]	.064	.141	.134	[-.110, .415]	-.024	-.083	.151	[-.383, .215]
Inattention and impulsivity *(mediator 1)*					.154	.028	.017	[-.001, .064]	.030	.008	.017	[-.023, .044]
SDQ *(moderator)*	.069	1.541	1.173	[-.518, 4.129]								
TBI x SDQ *(interaction effect)*	.195	8.801	3.353	[2.151, 15.078]								
Substance use *(mediator 2)*									.388	.610	.142	[.324, .886]
Conditional indirect effects									**B**		**SE**	**95% CI**
Via Inattention and impulsivity												
SDQ=0 *(low value)*									.002		.009	[-.007, .036]
SDQ=1 *(high value)*									.077		.169	[-.191, .524]
Via substance use									.086		.085	[-.057, .285]
Via both mediators, given values of SDQ												

SDQ=0 *(low value)*	.004	.009	[-.006, .035]
SDQ=1 *(high value)*	.153	.115	[.010, .498]
Conditional total effects			
SDQ=0 *(low value)*	.009	.139	[-.245, .294]
SDQ=1 *(high value)*	.233	.244	[-.146, .844]

Note. TBI = Traumatic Brain injury; SDQ = Strengths and Difficulties Questionnaire; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap sample.

Appendix C: Sensitivity analyses comparing the group with orthopaedic injury versus those with no history of any injury

Table 15: Results of Model 1 (basic mediation model).

Model fit: *CFI* =1.000, *TLI* =1.000, *RMSEA*= .000, 90% CI =.000, .000.

	Inattention and impulsivity *(Mediator)*				Antisocial behaviour *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct effects								
Orthopaedic injury *(predictor)*	-.001	-.007	.177	[-.343, .346]	.023	.073	.058	[-.037, .197]
Inattention and impulsivity *(mediator)*					.164	.047	.013	[.025, .075]
Indirect effects								
Orthopaedic injury on antisocial behaviour through inattention and impulsivity					.000	.000	.009	[-.017, .017]
Total effects								
Orthopaedic injury on antisocial behaviour					.023	.072	.058	[-.038, .186]

Note. TBI = Traumatic Brain injury; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap samples.

Table 16: Results of Model 2 (moderated mediation model).

Model fit: *CFI* =1.000, *TLI* =1.062, *RMSEA*= .000, 90% CI =.000, .048.

	Inattention and impulsivity *(Mediator)*				**Antisocial behaviour** *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct and interaction effects								
Orthopaedic injury *(predictor)*	.011	.124	.186	[-.225, .501]	.026	.083	.061	[-.033, .208]
Inattention and impulsivity *(mediator)*					.162	.046	.014	[.023, .076]
SDQ *(moderator)*		3.380	.548	[2.314, 4.475]				
Orthopaedic injury x SDQ *(interaction effect)*		-1.671	1.314	[-4.077, 1.138]				
Conditional indirect effects at different values of SDQ					**B**	**SE**		**95% CI**
SDQ=0 *(low value)*					.006	.009		[-.010, .027]
SDQ=1 *(high value)*					-.071	.067		[-.235, .038]
Total effects at different values of SDQ								
SDQ=0 *(low value)*					.089	.061		[-.028, .211]
SDQ=1 *(high value)*					.012	.093		[-.189, .181]

Note. TBI = Traumatic Brain injury; SDQ = Strengths and Difficulties Questionnaire; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap sample.

Table 17: Results of Model 3 (moderated mediation model).

Model fit: *CFI*=1.000, *TLI*=1.031, *RMSEA*=.000, 90% CI =.000, .015.

	Inattention and impulsivity *(Mediator)*				**Antisocial behaviour** *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct and interaction effects								
Orthopaedic injury *(predictor)*	.003	.031	.225	[-.399, .474]	.026	.082	.061	[-.036, .204]
Inattention and impulsivity *(mediator)*					.149	.042	.013	[.020, .071]
FAI *(moderator)*	.060	.151	.067	[.023, .286]				
Orthopaedic injury x FAI *(interaction effect)*	.003	.014	.123	[-.223, .265]				
Conditional indirect effects at different values of SDQ						**B**	**SE**	**95% CI**
FAI=0 *(low value)*						.001	.010	[-.017, .022]
FAI=1 *(medium value)*						.002	.008	[-.013, .020]
FAI=3 *(high value)*						.003	.013	[-.022, .031]
Total effects at different values of SDQ								
FAI=0 *(low value)*						.083	.061	[-.033, .207]
FAI=1 *(medium value)*						.083	.061	[-.033, .205]
FAI=3 *(high value)*						.085	.061	[-.034, .205]

Note. TBI = Traumatic Brain injury; FAI = Family Adversity Index; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap sample.

Table 18: Results of Model 4 (moderated mediation model).

Model fit: *CFI*=.850, *TLI*=.363, *RMSEA*=.080, 90% CI =.055, .108.

	Inattention and impulsivity *(Mediator)*				**Antisocial behaviour** *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct and interaction effects								
Orthopaedic injury *(predictor)*	.007	.057	.288	[-.491, .633]	.030	.070	.087	[-.086, .259]
Executive dysfunctions symptoms *(mediator)*					.166	.043	.018	[-.491, .633]
SDQ *(moderator on pathway a)*	.157	2.819	1.000	[1.061, 5.021]				
Orthopaedic injury x SDQ *(interaction effect)*	-.018	-.638	1.514	[-3.735, 2.194]				
Substance use *(moderator on pathway b)*					.127	.131	.065	[.017, .273]
Inattention and impulsivity x Substance use *(interaction effect)*					.235	.048	.028	[-.006, .103]
Conditional indirect effects at different values of moderators					**B**		**SE**	**95% CI**
SDQ=0								
Substance use=-.844 *(low value)*					.000		.005	[-.008, .012]
Substance use=-.538 *(medium value)*					.001		.006	[-.009, .017]
Substance use=.205 *(high value)*					.003		.017	[-.024, .045]
SDQ=1								
Substance use=-.844 *(low value)*					-.002		.026	[-.071, .039]
Substance use=-.538 *(medium value)*					-.010		.034	[-.120, .033]
Substance use=.205 *(high value)*					-.031		.087	[-.267, .101]

Conditional total effects at different values of moderators			
SDQ=0			
Substance use=-.844 *(low value)*	.070	.087	[-.085, .258]
Substance use=-.538 *(medium value)*	.071	.087	[-.085, .258]
Substance use=.205 *(high value)*	.073	.088	[-.086, .263]
SDQ=1			
Substance use=-.844 *(low value)*	.068	.093	[-.099, .264]
Substance use=-.538 *(medium value)*	.060	.095	[-.118, .255]
Substance use=.205 *(high value)*	.039	.124	[-.210, .272]

Note. TBI = Traumatic Brain injury; SDQ = Strengths and Difficulties Questionnaire; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap sample.

Table 19: Results of Model 5 (moderated mediation model).

Model fit: *CFI*=.059, *TLI*=-3.000, *RMSEA*=.747, 90% CI =.732, .763.

	Inattention and impulsivity *(Mediator)*				**Antisocial behaviour** *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct and interaction effects								
Orthopaedic injury *(predictor)*	.019	.214	.200	[-.167, .623]	.014	.049	.069	[-.083, .186]
Inattention and impulsivity *(mediator)*					-.340	-.110	.035	[-.186, -.047]
SDQ *(moderator on pathway a)*	.175	3.653	.609	[2.522, 4.921]				
Orthopaedic injury x SDQ *(interaction effect)*	-.027	-1.357	1.539	[-4.027, 1.947]				
Parental monitoring *(moderator on pathway b)*					.022	.015	.016	[-.017, .047]
Inattention and impulsivity x Parental monitoring *(interaction effect)*					.522	.022	.006	[.011, .035]
Conditional indirect effects at different values of moderators					**B**		**SE**	**95% CI**
SDQ=0								
Parental monitoring=4 *(low value)*					-.005		.006	[-.024, .002]
Parental monitoring=6 *(medium value)*					.005		.005	[-.002, .018]
Parental monitoring=8 *(high value)*					.014		.014	[-.009, .046]
SDQ=1								
Parental monitoring=4 *(low value)*					.026		.042	[-.030, .146]
Parental monitoring=6 *(medium value)*					-.024		.037	[-.115, .036]

Parental monitoring=8 *(high value)*	-.074	.105	[-.294, .123]
Conditional total effects at different values of moderators			
SDQ=0			
Parental monitoring=4 *(low value)*	.044	.069	[-.090, .182]
Parental monitoring=6 *(medium value)*	.053	.069	[-.078, .192]
Parental monitoring=8 *(high value)*	.063	.070	[-.069, .204]
SDQ=1			
Parental monitoring=4 *(low value)*	.074	.081	[-.076, .242]
Parental monitoring=6 *(medium value)*	.024	.079	[-.130, .177]
Parental monitoring=8 *(high value)*	-.026	.127	[-.283, .219]

Note. TBI = Traumatic Brain injury; SDQ = Strengths and Difficulties Questionnaire; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap sample.

Table 20: Results of Model 6 (moderated serial mediation model).

Model fit: *CFI*=.964, *TLI*=.893, *RMSEA*=.023, 90% CI =.014, .034.

	Inattention and impulsivity *(Mediator 1)*				**Substance use** *(Mediator 2)*				**Antisocial behaviour** *(Dependent variable)*			
	β	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**	**β**	**B**	**SE**	**95% CI**
Direct and interaction effects												
Orthopaedic injury *(predictor)*	.011	.122	.188	[-.233, .505]	-.001	-.002	.056	[-.111, .108]	.024	.074	.061	[-.043, .194]
Inattention and impulsivity *(mediator 1)*					.164	.033	.011	[.014, .055]	.102	.029	.013	[.006, .057]
SDQ *(moderator)*	.163	3.375	.553	[2.325, 4.483]								
Orthopaedic injury x SDQ *(interaction effect)*	-.035	-1.706	1.296	[-4.050, 1.053]								
Substance use *(mediator 2)*									.345	.480	.076	[.335, .633]
Conditional indirect effects										**B**	**SE**	**95% CI**
Via inattention and impulsivity												
SDQ=0 *(low value)*										.004	.006	[-.006, .020]
SDQ=1 *(high value)*										-.046	.047	[-.181, .016]
Via substance use										-.001	.027	[-.055, .054]
Via both mediators, given values of SDQ												
SDQ=0 *(low value)*										.002	.003	[-.003, .010]
SDQ=1 *(high value)*										-.025	.024	[-.084, .013]

Conditional total effects			
SDQ=0 *(low value)*	.079	.060	[-.036, .201]
SDQ=1 *(high value)*	.003	.092	[-.195, .168]

Note. TBI = Traumatic Brain injury; SDQ = Strengths and Difficulties Questionnaire; β = standardized coefficient; B = unstandardized coefficient; CI = confidence interval; SE=standard error (unstandardized). Standard errors and 95% CIs are bootstrapped estimates based on 10,000 bootstrap sample.